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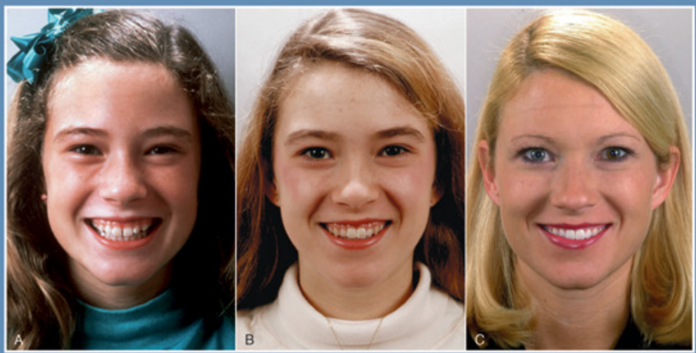

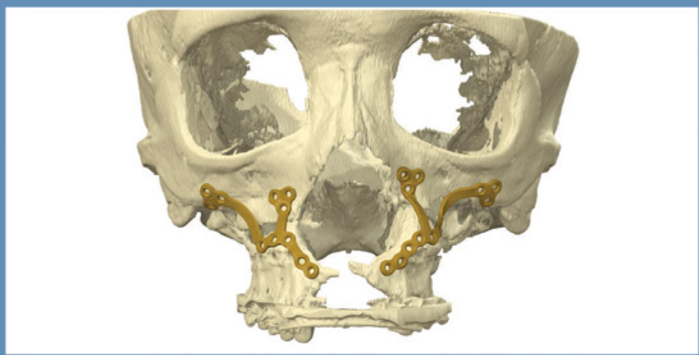

SEVENTH
EDITION

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Contemporary Orthodontics

Seventh Edition

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Preface

This is the seventh edition of *Contemporary Orthodontics*. The first edition was published in 1986—nearly 40 years ago. We have tried to keep the focus the same. Dental students can learn basic growth and development, the etiology of orthodontic problems, and some treatment methods. New residents can be introduced to orthodontic dilemmas, perspectives, and treatment alternatives based on evidence. Practitioners can be updated and reflect on their orthodontic care, the evolving orthodontic discipline, and the treatment provided by colleagues.

What is new? Lots! We have a smaller author team because of the death of Bill Proffit. Bill created the foundation of this book when he was building the self-instruction orthodontic curriculum at the University of Florida and then enhanced it at the University of North Carolina starting in 1975. Over the next decade, we joined him as faculty and residents and later colleagues. We worked together on more projects than just this book. His strength was to organize and simplify a problem, its essence, and solutions. He was trained as a physiologist as well and liked gadgets and quantifiable results but was not averse to speculating on data. He was a relentless worker, motivator, and thinker. Our part in this partnership, which continues in his stead, was and is to explore, reflect, challenge, distill, and make orthodontics available to all with an interest and desire to learn. Marked by Bill as we were, we strive to make sense of this exciting discipline that still has more questions than answers.

In addition to new topics and images, we have added more contributors: Matthew Larson, Regina Blevins, Kaitrin Kramer, Benjamin T. Pliska, Ching-Chang Ko, Toru Deguchi, and Benjamin W. Fields, whose contributions are listed in the Table of Contents and the chapter headings. They have helped us add:

- An artificial intelligence perspective and potential.
- Data evaluation and priority.
- A voice for standard metrics in orthodontic research.
- Social and behavioral problems related to orthodontics.
- An overview of common craniofacial problems.
- Obstructive sleep apnea as a diagnostic and treatment-related issue.
- Revised diagnosis and treatment planning with more specific guidance.
- Systematic diagnostic checklists.
- Updated informed consent as understanding.
- Clear tray aligners and hybrid treatment for adolescents and adults.
- Expanded biomechanics related to clear tray aligners.
- More discussion of skeletal anchorage options and applications.
- 3D options for orthognathic surgery planning.

This is a book that attempts to rely on evidence and is moderately referenced, but more than previous editions. We intend that the conclusions are evidence informed. With the remembrance of Bill and his guidance and in the traditions set in previous editions, we carry on.

Henry W. Fields, Jr.
Brent E. Larson
David M. Sarver

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We thank those who provided resources and aid not recognized in print. The residents of the Orthodontic classes of 2024 at the Ohio State University College of Dentistry Division of Orthodontics and the University of Minnesota Division of Orthodontics, and the Division of Pediatric Dentistry Class of 2024 at Ohio State University College of Dentistry and Nationwide Children's Hospital supplied critiques, ideas, and images to support this publication.

Our new authors, who are acknowledged in the Table of Contents and chapter headings included, Drs. Ching-Chang Ko, Kaitrin Kramer, Benjamin W. Fields, Benjamin T. Pliska, Matthew Larson, Toru Deguchi, and Regina Blevins. They expanded our breadth and depth of knowledge. As always, Dr. Tim Shaughnessy provided sources of images and cases, and Dr. Joen Iannucci provided radiologic support information and advice.

Dr. Tessa Streeter proofread sections of the text in an extremely thorough and timely manner to enhance our responses to the publisher.

We appreciate the support of Elsevier, who has worked with us for seven editions.

Thank you.

Henry W. Fields, Jr.
Brent E. Larson
David M. Sarver

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SECTION 1

The Orthodontic Problem

This section of the book addresses important questions that are the intellectual and scientific background for the practice of orthodontics:

Why do we provide orthodontic treatment?
Who needs treatment?
How do people benefit from it?
How prevalent are orthodontic problems?
How do children, adolescents and adults grow and develop
How are these problems related to growth of the head and face?
How are these problems related to eruption of the teeth?
Can we identify the etiology of these orthodontic problems?
How do we make decisions about treatment based on the evidence?

You need to consider the answers to these questions before you can appropriately diagnose orthodontic problems, plan the treatment that will provide maximum benefit to the patient, and carry out that treatment. The answers, to the best of our ability to provide them now, are in chapters1–5.

1

Malocclusion and Dentofacial Abnormalities in Contemporary Society

HENRY W. FIELDS JR., WILLIAM R. PROFFIT AND CHING-CHANG KO

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The Changing Goals of Orthodontic Treatment

The Development of Orthodontics

Crowded, irregular, and protruding teeth have been a problem for some individuals since antiquity and attempts to correct this disorder go back at least to 1000 BC. Primitive (and surprisingly well-designed) orthodontic appliances have been found in both Greek and Etruscan materials.[1] As dentistry developed in the 18th and 19th centuries, a number of devices for the "regulation" of the teeth were described by various authors and apparently used sporadically by the dentists of that era.

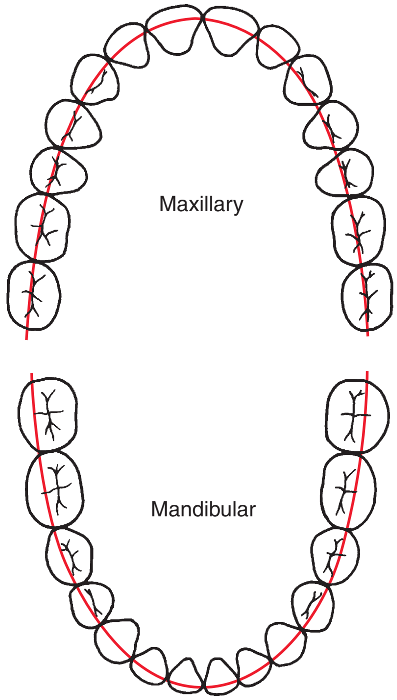
After 1850 the first texts that systematically described orthodontics appeared, the most notable being Norman Kingsley's *Oral Deformities*.[2] Kingsley, who had a tremendous influence on American dentistry in the latter half of the 19th century, was among the first to use extraoral force to correct protruding teeth. He was also a pioneer in the treatment of cleft palate and related problems. Kingsley also wrote about the importance of the artistic form of the face and recognized the importance of facial esthetics.

Despite the contributions of Kingsley and his contemporaries, the emphasis in orthodontics remained the alignment of the teeth and the correction of facial proportions. Little attention was paid to bite relationships, and because it was common practice to remove teeth for many dental problems, extractions for crowding or malalignment were frequent. In an era when an intact dentition was a rarity, the details of occlusal relationships were considered unimportant.

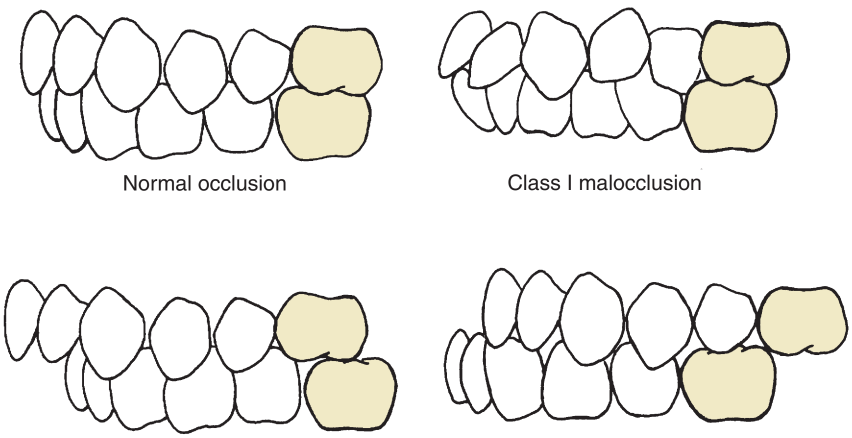
To make good prosthetic replacement teeth, it was necessary to develop a concept of occlusion, and this occurred in the late 1800s. As the concepts of prosthetic occlusion developed and were refined, it was natural to extend this to the natural dentition. Edward H. Angle (Fig. 1.1), whose influence began to be felt about 1890, can be credited with much of the development of a concept of occlusion in the natural dentition. Angle's original interest was in prosthodontics, and he taught in that department in dental schools in Pennsylvania and Minnesota in the 1880s. His increasing interest in dental occlusion and in the treatment necessary to obtain normal occlusion led directly to his development of orthodontics as a specialty, with himself as the "father of modern orthodontics."

Angle's classification of malocclusion in the 1890s was an important step in the development of orthodontics because it not only subdivided major types of malocclusion but also included the first clear and simple definition of normal occlusion in the natural dentition. Angle's postulate was that the upper first molars were the key to occlusion and that the upper and lower molars should be related so that the mesiobuccal cusp of the upper molar occludes in the buccal groove of the lower molar. If the teeth were arranged on a smoothly curving line of occlusion (Fig. 1.2) and this molar relationship existed (Fig. 1.3), then normal occlusion

• **Fig. 1.1** Edward H. Angle in his 50s, as the proprietor of the Angle School of Orthodontia. After establishing himself as the first dental specialist, Angle operated proprietary orthodontic schools from 1905 to 1928 in St. Louis, Missouri; New London, Connecticut; and Pasadena, California, in which many of the pioneer American orthodontists were trained.



• **Fig. 1.2** The line of occlusion is a smooth (catenary) curve passing through the central fossa of each upper molar and across the cingulum of the upper canine and incisor teeth. The same line runs along the buccal cusps and incisal edges of the lower teeth, thus specifying the occlusal as well as interarch relationships once the molar position is established.



• **Fig. 1.3** Normal occlusion and malocclusion classes as specified by Angle. This classification was quickly and widely adopted early in the 20th century. It is incorporated within all contemporary descriptive and classification schemes.

would result.[3] This statement, which 100 years of experience has proved to be correct except when there are aberrations in the size of teeth, brilliantly simplified normal occlusion.

Angle then described three classes of *malocclusion*, based on the occlusal relationships of the first molars:

- Class I: Normal relationship of the molars, but line of occlusion incorrect because of malposed teeth, rotations, or other causes
- Class II: Lower molar distally positioned relative to upper molar, line of occlusion not specified
- Class III: Lower molar mesially positioned relative to upper molar, line of occlusion not specified

Note that the Angle classification has four classes: normal occlusion, Class I malocclusion, Class II malocclusion, and Class III malocclusion (see Fig. 1.3). Normal occlusion and Class I malocclusion share the same molar relationship but differ in the arrangement of the teeth relative to the line of occlusion. The line of occlusion may or may not be correct in Class II and Class III malocclusion.

With the establishment of a concept of normal occlusion and a classification scheme that incorporated the line of occlusion, by the early 1900s orthodontics was no longer just the alignment of irregular teeth. Instead, it had evolved into the treatment of malocclusion, defined as any deviation from the ideal occlusal scheme described by Angle. Because precisely defined relationships required a full complement of teeth in both arches, maintaining an intact dentition became an important goal of orthodontic treatment. Angle and his followers strongly opposed extraction for orthodontic purposes. With the emphasis on dental occlusion that followed, however, less attention came to be paid to facial proportions and esthetics. Angle abandoned extraoral force because he decided this was not necessary to achieve proper occlusal relationships. He solved the problem of dental and facial appearance by simply postulating that the best esthetics always were achieved when the patient had ideal occlusion.

As time passed, it became clear that even an excellent occlusion was unsatisfactory if it was achieved at the expense of proper facial proportions. Calvin Case was an advocate of carefully considering protrusion and extraction treatment in order to achieve the best dentofacial harmony for the individual. With that he also realized that tipping and root movement could also bring different esthetic results.[4]

Not only had some realized that the teeth needed to be related to the face, Dr. Paul Simon believed that the dentition was invariably related to the skull. His theory that the canine was related to the orbital plane was examined by Broadbent using skulls and a craniostat. It was found to be too variable to be of use as an orthodontic landmark for the dentition.[5] But, as orthodontics was developing as a profession some were examining more than just the alignment of the teeth, with the addition of lip protrusion and facial esthetics.

Not only were there esthetic problems, it often proved impossible to maintain an occlusal relationship achieved by prolonged use of heavy elastics to pull the teeth together as Angle and his followers had suggested. So, early practitioners discovered retention issues as well.

Under the leadership of Charles Tweed in the United States and Raymond Begg in Australia (both of whom had studied with Angle), extraction of teeth was reintroduced into orthodontics in the 1940s and 1950s to enhance facial esthetics and achieve better stability of the occlusal relationships.

Cephalometric radiography, which enabled orthodontists to measure the changes in tooth and jaw positions produced by growth and treatment, came into widespread use after World War II. These radiographs made it clear that many Class II and Class III malocclusions resulted from faulty jaw relationships, not just malposed teeth. By use of cephalometrics, it was also possible to see that jaw growth could be altered by orthodontic treatment. In Europe, the method of “functional jaw orthopedics” was developed to enhance growth changes, while in the United States, extraoral force came to be used for this purpose. At present, both functional and extraoral appliances are used internationally to control and modify growth and form. Obtaining correct or at least improved jaw relationships became a goal of treatment by the mid-20th century.

The changes in the goals of orthodontic treatment, which now focus on facial proportions and the impact of the dentition on facial appearance, have been codified in the form of the soft tissue paradigm.[6]

Modern Treatment Goals: The Soft Tissue Paradigm

A paradigm can be defined as “a set of shared beliefs and assumptions that represent the conceptual foundation of an area of science or clinical practice.” The soft tissue paradigm states that both the goals and limitations of modern orthodontic and orthognathic treatment are determined by the soft tissues of the face, not by the teeth and bones. This reorientation of orthodontics away from the Angle paradigm that dominated the 20th century is most easily understood by comparing treatment goals, diagnostic emphasis, and treatment approach in the two paradigms (Table 1.1). With the soft tissue paradigm, the increased focus on clinical examination rather than examination of dental casts and radiographs leads to a different approach to obtaining important diagnostic information, and that information is used to develop treatment plans that would not have been considered without it.

More specifically, what difference does the soft tissue paradigm make in planning treatment? There are several major effects:

1. The primary goal of treatment becomes esthetic and proportional soft tissue relationships and adaptations, not Angle's ideal occlusion. This broader goal is not incompatible with Angle's ideal occlusion, but it acknowledges that to provide maximum benefit for the patient, ideal occlusion (in terms of a nonextraction ideally aligned dentition) cannot always be the major focus of a treatment plan. Soft tissue relationships, both the 3D proportions of the facial soft tissue and the protrusive/retrusive relationships of the dentition to the lips and face, are the major determinants of facial appearance. Soft tissue adaptations to the position of the teeth (or lack of it) determine whether the orthodontic result will be stable and esthetic. Dentofacial appearance affects social interactions, but also

TABLE 1.1 Angle Versus Soft Tissue Paradigms: A New Way of Looking at Treatment Goals

Parameter	Angle Paradigm	Soft Tissue Paradigm
Primary treatment goal	Ideal dental occlusion	Normal soft tissue proportions and adaptations
Secondary goal	Ideal jaw relationships	Functional occlusion
Hard and soft tissue relationships	Ideal hard tissue proportions produce ideal soft tissues	Ideal soft tissue proportions define ideal hard tissues
Diagnostic emphasis	Dental casts, cephalometric radiographs	Clinical examination of intraoral and facial soft tissues
Treatment approach	Obtain ideal dental and skeletal relationships, assume the soft tissues will be all right	Plan ideal soft tissue relationships and then place teeth and jaws as needed to achieve this
Function emphasis	TMJ in relation to dental occlusion	Soft tissue movement in relation to display of teeth
Stability of result	Related primarily to dental occlusion	Related primarily to soft tissue pressure and equilibrium effects

TMJ, Temporomandibular joint.

perceptions of individuals' ability to learn and be employed. Keeping this in mind while planning treatment is critically important for complete success.

2. The secondary goal of treatment becomes *functional occlusion*. What does that have to do with soft tissues? Temporomandibular (TM) dysfunction, to the extent that it relates to dental occlusion, is best thought of as the result of injury to the soft tissues around the temporomandibular joint (TMJ) caused by clenching and grinding the teeth, whatever the source of the injury and associated pain and dysfunction. Given that, an important goal of treatment is to arrange the occlusion to minimize the chance of injury. In this also, Angle's ideal occlusion is not incompatible with the broader goal, but deviations from the Angle ideal may provide greater benefit for some patients and should be considered when treatment is planned.
3. The thought process that goes into "solving the patient's problems" is reversed. In the past, the clinician's focus was on dental and skeletal relationships, with the tacit assumption that if these were correct, soft tissue relationships would take care of themselves. With the broader focus on facial and oral soft tissues, the thought process is to establish what these soft tissue relationships should be and then determine how the teeth and jaws would have to be arranged to meet the soft tissue goals. Why is this important in establishing the goals of treatment? It relates very much to why patients and parents seek orthodontic treatment and what they expect to gain from it. Most want an appearance so they can function as productive and self-assured members of society.

The following sections of this chapter provide some background on the prevalence of malocclusion, what we know about the need for treatment of malocclusion and dentofacial abnormalities, and how soft tissue considerations, as well as teeth and bone, affect both need and demand for orthodontic treatment. It must be kept in mind that orthodontics is shaped by biological, psychosocial, and cultural determinants. For that reason, when defining the goals of orthodontic treatment, one has to consider not only morphologic and functional factors, but a wide range of psychosocial and bioethical issues as well. All these topics are discussed in much greater detail in the following chapters on diagnosis, treatment planning, and treatment.

The Usual Orthodontic Problems: Epidemiology of Malocclusion

Angle's "normal occlusion" more properly should be considered the ideal without a facial context. In fact, perfectly interdigitating teeth arranged along a perfectly regular line of occlusion are quite rare. For many years, epidemiologic studies of malocclusion suffered from considerable disagreement among investigators about how much deviation from the ideal should be accepted within the bounds of normal. By the 1970s, a series of studies by public health or university groups in most developed countries provided a reasonably clear worldwide picture of the prevalence of the various types of malocclusion by degree of severity.

In the United States, two large-scale surveys carried out by the US Public Health Service (USPHS) covered children ages 6 to 11 years from 1963 to 1965 and youths ages 12 to 17 years in 1969 and 1970.[7,8] As part of a large-scale national survey of health care problems and needs in the United States in 1989 through 1994 (Third National Health and Nutrition Examination Survey [NHANES III]), estimates of malocclusion again were obtained. This study of 14,000 individuals was statistically designed to provide weighted estimates for approximately 150 million persons in the sampled racial or ethnic and age groups. The data provide reasonably current information for US children and youths and include the first good data set for malocclusion in adults, with separate estimates for the major racial (White and Black) and one ethnic group (Mexican American).[9]

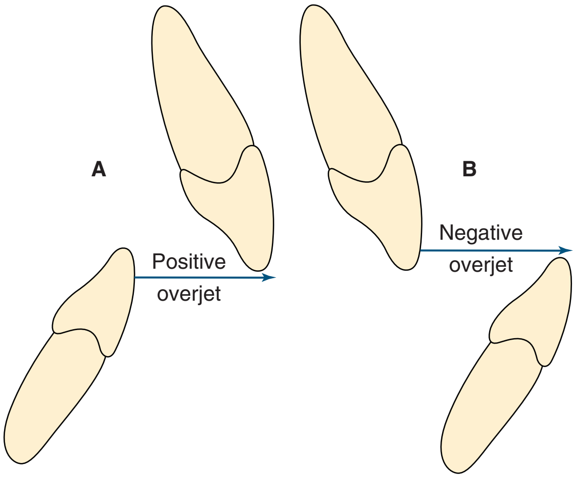
The characteristics of malocclusion evaluated in NHANES III included the irregularity index, which is a measure of incisor alignment (Fig. 1.4); the prevalence of midline diastema larger than 2 mm (Fig. 1.5); and the prevalence of posterior crossbite (Fig. 1.6). In addition, overjet (Fig. 1.7) and overbite or open bite (Fig. 1.8) were measured. Overjet reflects Angle's Class II and Class III molar relationships. Because overjet can be evaluated much more precisely than molar relationship in a clinical examination, molar relationship was not evaluated directly.

Data for these characteristics of malocclusion for the children (age 8–11), youth (12–17) and adults (18–50) are provided for the US population, taken from the NHANES III study, and are displayed graphically in Figs. 1.9–1.13.

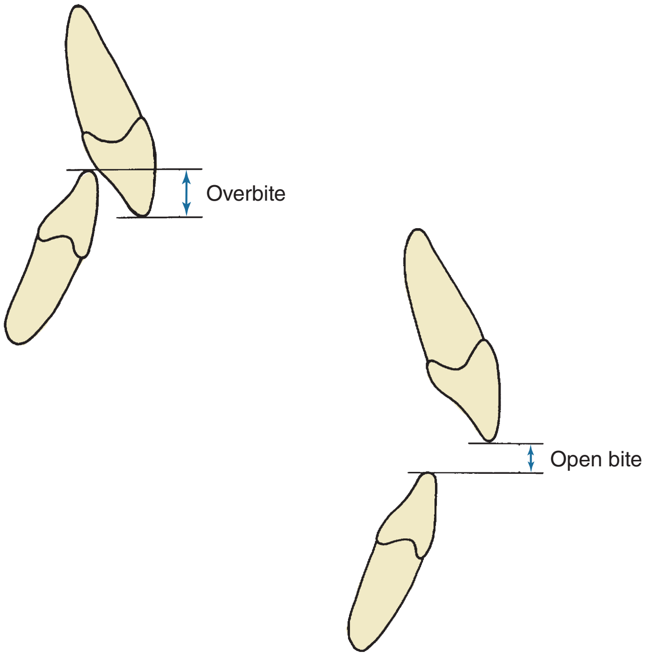
• **Fig. 1.4** Incisor irregularity usually is expressed as the irregularity index: the total of the millimeter distances from the anatomic contact point on each incisor tooth to the anatomic contact point that it should touch, as shown by the blue lines. For this patient, the irregularity index is 10 (mm).

• **Fig. 1.5** A space between adjacent teeth is called a *diastema*. A maxillary midline diastema is relatively common, especially during the mixed dentition in childhood, and disappears or decreases in width as the permanent canines erupt. Spontaneous correction of a childhood diastema is most likely when its width is less than 2 mm, so this patient is on the borderline and may need future treatment.

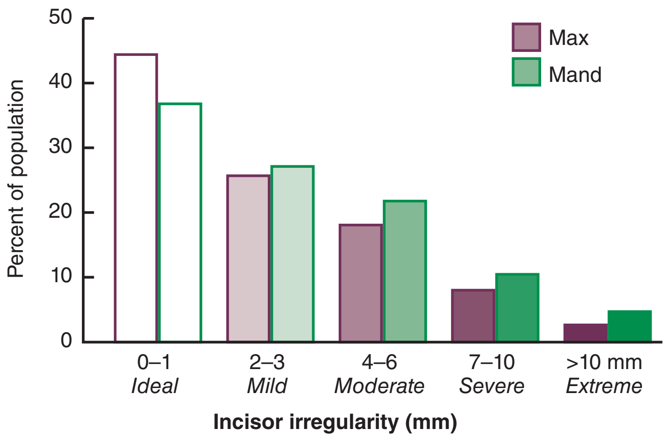
• **Fig. 1.6** Posterior crossbite exists when the maxillary posterior teeth are lingually positioned relative to the mandibular teeth, as in this patient. Posterior crossbite most often reflects a narrow maxillary dental arch but can arise from other causes. This patient also has a one-tooth anterior crossbite, with the lateral incisor trapped lingually.



• **Fig. 1.7** Overjet is defined as horizontal overlap of the incisors, measured from the facial surface of the most prominent mandibular incisor to the facial surface of the most prominent maxillary incisor. Normally the incisors are in or near contact, with the upper incisors ahead of the lower by only the thickness of their incisal edges (i.e., positive overjet of +2 to 3 mm). If the lower incisors are in front of the upper incisors, with overjet measured from the same facial surfaces, the condition is called *reverse or negative overjet* or *anterior crossbite*.



• **Fig. 1.8** Overbite is defined as the vertical overlap of the incisors. Normally, the lower incisal edges contact the lingual surface of the upper incisors at or above the cingulum (i.e., normally there is a 1- to 2-mm overbite). In open bite, there is no vertical overlap, and the vertical separation of the incisors is measured to quantify its severity and given a negative value of, say -3 mm.



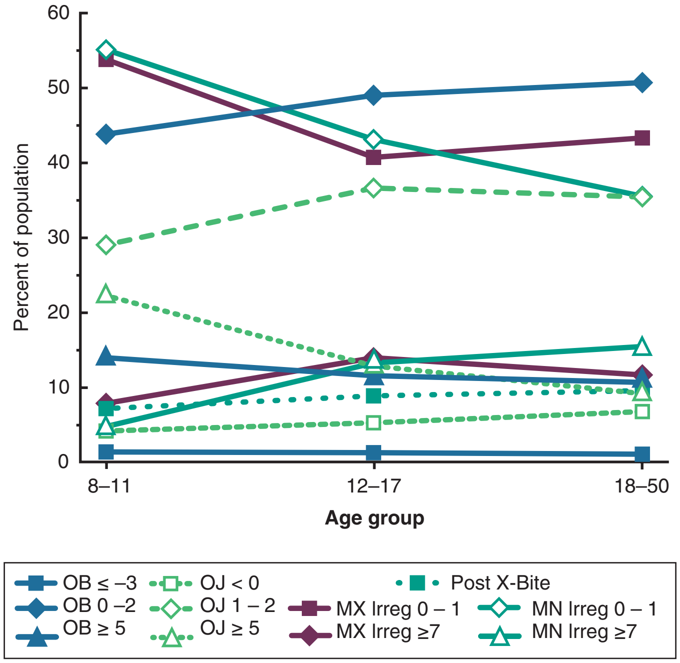
• **Fig. 1.9** Incisor irregularity in the US population, 1989–94. Of the population, 20%–25% have moderately irregular (usually crowded) incisors, and nearly 15% have severe or extreme irregularity. Note that irregularity in the lower arch is more prevalent at any degree of severity.

Note in Fig. 1.9 incisor irregularity is common and usually more so in the lower arch. Approximately 15% have severe or extreme alignment problems. Major arch expansion or extraction of some teeth would be necessary to align them.

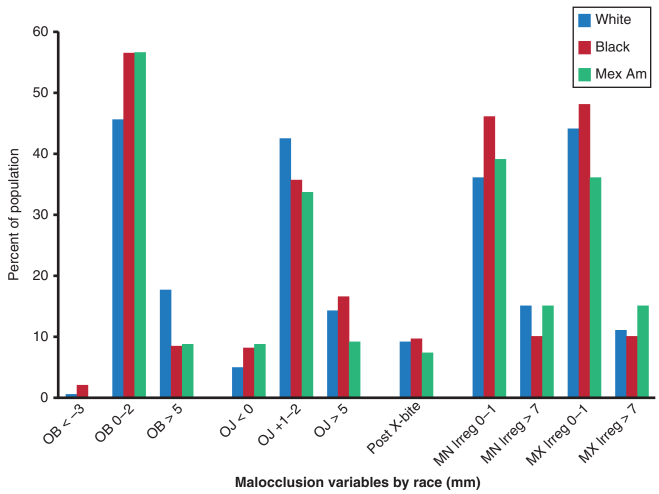
In the age 8 to 11 group, just over half of US children have well-aligned incisors. The rest have varying degrees of malalignment and crowding (Fig. 1.10). The percentage with excellent alignment decreases in the age 12 to 17 group as the remaining permanent teeth erupt, then remains essentially stable in the upper arch but worsens in the lower arch for adults.

Racial and ethnic differences are apparent from the data. Mexican Americans are more likely to have severe maxillary crowding, but they and Whites have the most severe mandibular crowding (Fig. 1.11). A midline diastema larger than 2 mm is present in 26% of US children. This amount of space can be an esthetic problem and can cause space problems with eruption of the lateral incisors. Although this space tends to close, over 6% of youths and adults still have a noticeable diastema that compromises the appearance of the smile (see Fig. 1.5). Blacks are more than twice as likely to have a midline diastema as Whites or Mexican American.

Occlusal relationships must be considered in all planes of space. Lingual posterior crossbite (i.e., upper teeth lingual to lower teeth) is the major deviation from the normal transverse or coronal dental relationship. According to the NHANES III data,[9] it occurs in 7% to 8% of the US population. Posterior crossbites are near 7% in childhood, 8% in youth and 9% in adults (see Fig. 1.10).



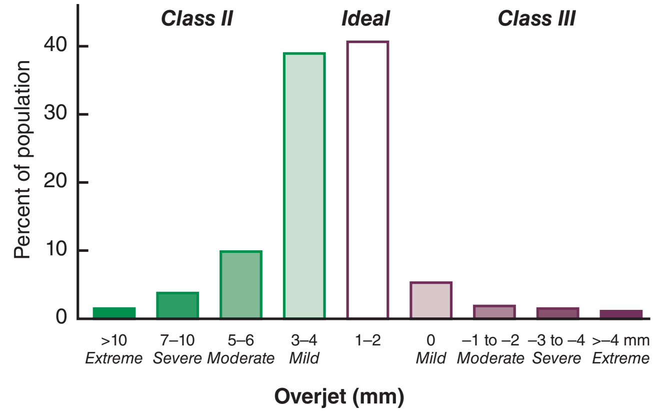
• **Fig. 1.10** Changes in the prevalence of types of malocclusion from childhood to adult life, United States, 1989–94. Note the increase in incisor irregularity, decrease in severe overjet and increase in negative overjet as children mature, both of which are related to more mandibular than maxillary growth. Posterior crossbites gradually increase with maturity. Open bites appear to be stable in number, while normal overbites increase and deep overbites decrease.



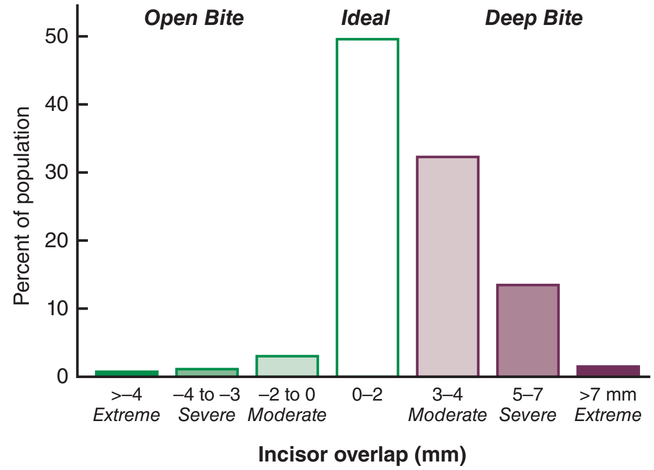
• **Fig. 1.11** Incisor irregularity by racial or ethnic groups. The percentage of the Mexican American population with severe crowding is equal or greater than the other two groups for both the maxilla and mandible followed by Whites. ideal alignment is lowest for Whites in the mandible and Mexican Americans in the maxilla. This may reflect the low number of Mexican Americans with orthodontic treatment at the time of the NHANES III survey. Posterior crossbites are similar in all groups. Increased overjet is more frequent in Blacks and negative overjet is more frequent in Blacks and Mexican Americans. Vertically, deep bites are a common White trait and open bites more common in Blacks.

Posterior crossbite differs little among the racial and ethnic groups (see Fig. 1.11).

Normal overjet is 2 mm. Increased positive overjet or reverse overjet (negative) indicates anteroposterior deviations in the



• **Fig. 1.12** Overjet (Class II) and reverse overjet (Class III) in the US population, 1989–94. Only one-third of the population have ideal anteroposterior incisor relationships, but overjet is only moderately increased in another one-third. Increased overjet accompanying Class II malocclusion is much more prevalent than reverse overjet accompanying Class III.



• **Fig. 1.13** Open bite and deep bite relationships in the US population, 1989–94. Half the population have an ideal vertical relationship of the incisors. Deep bite is much more prevalent than open bite, but vertical relationships vary greatly among racial groups (Fig. 1.11).

Class II or Class III direction, respectively, with Class III being much less prevalent (Fig. 1.12). Overjet of 5 mm or more, suggesting Angle's Class II malocclusion, occurs in 22% of children, 13% of youths, and 9% of adult, while 0 mm or <0 mm overjet increases over time (Fig. 1.10). This reflects the greater postnatal growth of the mandible than the maxilla, which is discussed in Chapter 2. Class II problems are more prevalent in Black and White groups and Class III problems are more prevalent in Mexican American and Black groups (Fig. 1.11).

Vertical deviations from the ideal overbite of 2 mm are frequent (Fig. 1.13). More people in the US have deep bites than open bites. Open bites appear to remain stable, while deep bites become less severe (see Fig. 1.10). There are striking differences between the racial or ethnic groups in vertical dental relationships. Severe deep bite is nearly twice as prevalent in Whites as Blacks or Mexican Americans, whereas open bite of greate than 3 mm is more prevalent in Blacks (see Fig. 1.11). This almost surely reflects the slightly different craniofacial proportions of the Black population groups (see Chapter 5 for a more complete discussion). In contrast to the higher prevalence of anteroposterior problems, vertical problems are less prevalent in Latinx than either Blacks or Whites.

From the survey data, it is interesting to calculate the percentage of American children and youths who would clearly fall into Angle's Class I, II and III groups. From this perspective, 30+% at most have Angle's normal occlusion. Class II malocclusions (approximately 15-20+%); and Class III (less than ~5%). The remainder are variations of antero-posterior discrepancies.

Differences in malocclusion characteristics between the United States and other countries would be expected because of differences in racial and ethnic composition. Although the available data are not as extensive as for American populations, it seems clear that Class II problems are most prevalent in Whites of northern European descent (for instance, 25% of children in Denmark are reported to have Class II malocclusion), whereas Class III problems are most prevalent in Asian populations (3%–5% in Japan, nearly 2% in China), with another 2% to 3% pseudo–Class III (i.e., shifting into anterior crossbite because of incisor interferences). African populations are by no means homogenous, but from the differences found in the United States between Blacks and Whites, it seems likely that Class III and open bite are more frequent in African than European populations and deep bite less frequent.

Why Is Malocclusion So Prevalent?

Crowded and irregular teeth now occur in a majority of the population; skeletal remains indicate that this was unusual until relatively recently, although not unknown (Fig. 1.14). Because the mandible tends to become separated from the rest of the skull when long-buried skeletal remains are unearthed, it is easier to be sure what has happened to alignment of teeth than to occlusal relationships. The skeletal remains suggest that all members of a group might tend toward a Class III or, less commonly, a Class II jaw relationship. Similar findings are noted in present population groups (crowding and malalignment of teeth) that are uncommon in ancient skeletal remains. The majority of the group may have mild anteroposterior or transverse discrepancies, as in the Class III tendency of South Pacific islanders[10] and buccal crossbite (X-occlusion) in aboriginal people of Australia.[11]"

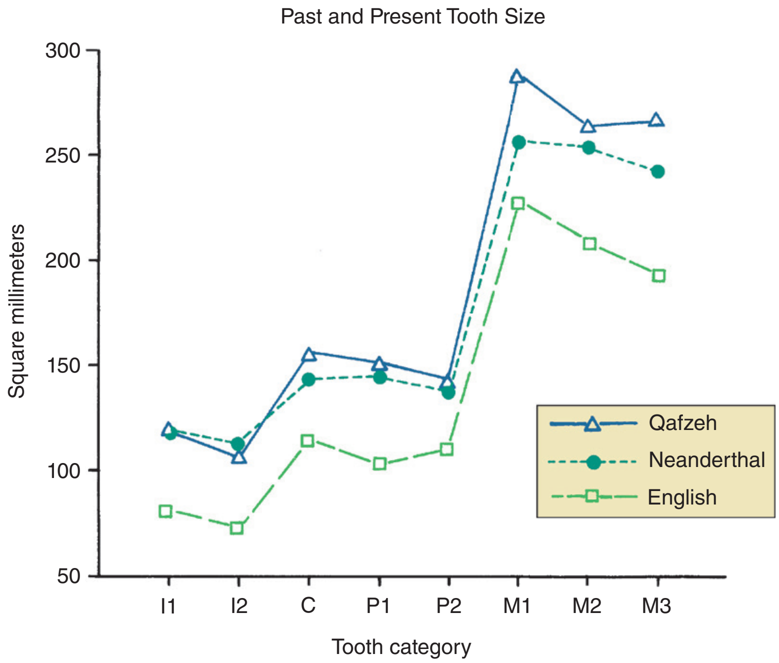
• **Fig. 1.14** Mandibular dental arches from specimens from the Krapina cave in Yugoslavia, estimated to be approximately 100,000 years old. Note the excellent alignment in this specimen. Near-perfect alignment or minimal crowding was the usual finding in this group. (From Wolpoff WH. *Paleoanthropology*. Alfred A Knopf; 1998.)

Although 1000 years is a long time relative to a single human life, it is a very short time from an evolutionary perspective. The fossil record documents evolutionary trends over many thousands of years that affect the present dentition, including a decrease in the size of individual teeth, in the number of the teeth, and in the size of the jaws. For example, there has been a steady reduction in the size of both anterior and posterior teeth over at least the last 100,000 years (Fig. 1.15). The number of teeth in the dentition of higher primates has been reduced from the usual mammalian pattern (Fig. 1.16). The third incisor and third premolar have disappeared, as has the fourth molar. At present, the human third molar, second premolar, and second incisor often fail to develop, which indicates that these teeth may be on their way out. Compared with other primates, modern humans have quite underdeveloped jaws.

It is easy to see that the progressive reduction in jaw size, if not well matched to a decrease in tooth size and number, could lead to crowding and malalignment. It is less easy to see why dental crowding should have increased quite recently, but this seems to have paralleled the transition from primitive agricultural to modern urbanized societies. Cardiovascular disease and related health problems appear rapidly when a previously unaffected population group leaves agrarian life for the city and civilization. High blood pressure, heart disease, diabetes, and several other medical problems are so much more prevalent in developed than underdeveloped countries that they have been labeled "diseases of civilization."

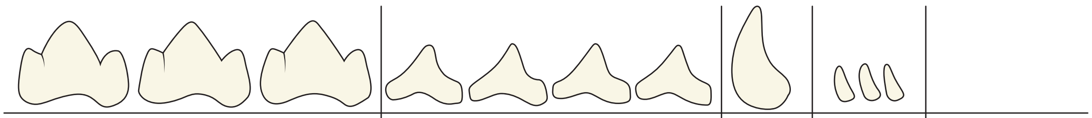
There is some evidence that malocclusion increases within well-defined populations after a transition from rural villages to the city. Corruccini, for instance, reported a higher prevalence of crowding, posterior crossbite, and buccal segment discrepancy in urbanized youths compared with rural Punjabi youths of northern India.[12] One can argue that malocclusion is another condition made worse by the changing conditions of modern life, perhaps resulting in part from less use of the masticatory apparatus with softer foods now. Under primitive conditions, of course, excellent function of the jaws and teeth was an important predictor of the ability to survive and reproduce. A capable masticatory apparatus was essential to deal with uncooked or partially cooked meat and plant foods. Watching an Australian aboriginal man using every muscle of his upper body to tear off a piece of kangaroo flesh from the barely cooked animal, for instance, makes one appreciate the decrease in demand on the masticatory apparatus that has accompanied civilization (Fig. 1.17). An interesting proposal by anthropologists is that the introduction of cooking, so that it did not take as much effort and energy to masticate food, was the key to the development of the larger human brain. Without cooked food, it would not have been possible to meet the energy demand of the enlarging brain. With it, excess energy is available for brain development and robust jaws are unnecessary.[13]

Determining whether changes in jaw function have increased the prevalence of malocclusion is complicated by the fact that both dental caries and periodontal disease, which are rare on the primitive diet, appear rapidly when the diet changes. The resulting dental pathology can make it difficult to establish what the occlusion might have been in the absence of early loss of teeth,



• **Fig. 1.15** The generalized decline in the size of human teeth can be seen by comparing tooth sizes from the anthropologic site at Qafzeh, dated 100,000 years ago; Neanderthal teeth, 10,000 years ago; and modern human populations. (Redrawn from Kelly MA, Larsen CS, eds. *Advances in Dental Anthropology*. Wiley-Liss; 1991.)

Molars (M)	Premolars (PM)	Canines (C)	Incisors (I)	



M-3	PM-4	C-1	I- 3	Basic mammalian
M-3	PM-3	C-1	I- 2	Prosimian
M-3	PM-2	C-1	I- 2	Higher ape
M-3 (2)	PM-(1-2)	C-1	I- (1-2)	Man

• **Fig. 1.16** Reduction in the number of teeth has been a feature of primate evolution. In the present human population, third molars are so frequently missing that it appears a further reduction is in progress, and the relatively high prevalence of missing maxillary lateral incisors and mandibular second premolars suggests evolutionary pressure on these teeth.

• **Fig. 1.17** A frame from a 1960s movie of an Australian aboriginal man eating a kangaroo prepared in the traditional (barely cooked) fashion. Note the activity of muscles, not only in the facial region, but throughout the neck and shoulder girdle. (Courtesy M. J. Barrett.)

gingivitis, and periodontal breakdown. The increase in malocclusion in modern times certainly parallels the development of modern civilization, but a reduction in jaw size related to disuse atrophy is hard to document. Although it is difficult to know the precise cause of any specific malocclusion, we do know in general what the etiologic possibilities are, and these are discussed in some detail in Chapter 5.

What difference does it make if you have a malocclusion? Let's now consider the reasons for orthodontic treatment.

Who Needs Treatment?

Protruding, irregular, or maloccluded teeth have been reported to cause three types of problems for the patient: (1) social discrimination because of facial appearance; (2) problems with oral function, including difficulties in jaw movement (muscle incoordination or pain), temporomandibular dysfunction (TMD), and problems with mastication, swallowing, or speech; and (3) greater susceptibility to trauma and periodontal disease, or tooth decay. Below we examine the evidence.

Psychosocial Problems

A number of studies in recent years have confirmed what is intuitively obvious: that severe malocclusion is likely to be a social handicap. The usual caricature of an individual who is not too bright often includes protruding upper incisors. A witch not only rides a broom, she has a prominent lower jaw that would produce a Class III malocclusion. Simply revealing malaligned teeth can reduce facial attractiveness. Well-aligned teeth and a pleasing smile carry positive status at all social levels and ages, whereas irregular or protruding teeth carry negative social cues, imply reduced intelligence and ethical behavior.[14–16] Appearance can and does make a difference in teachers' expectations and therefore in student progress in school, in employability, and in competition for a mate. This places the concept of "handicapping malocclusion" in a broader and more important context. If the way you interact with other individuals is affected constantly by your teeth, your dental handicap is far from trivial. There is no doubt that social responses conditioned by the major deviations from the usual appearance of the face and teeth can severely affect quality of life and self-esteem in a way that compromises an individual's whole adaptation to life.[17]

It is interesting that psychic distress caused by dental or facial abnormalities is not directly proportional to the anatomic severity of the problem. An individual with anomalies (e.g., with a distorted nose and scarred lip after cleft lip or palate repair) can anticipate a consistently negative response.[18] An individual with an apparently less severe problem (e.g., a deficient chin or protruding maxillary incisors) is sometimes treated differently because of this, but sometimes not. It seems to be easier to cope with a defect if other people's responses to it are consistent than if they are not. Unpredictable responses produce anxiety and can have strong deleterious effects.[19] The impact of a physical abnormality on an individual also will be strongly influenced by that person's self-esteem. The result is that the same degree of anatomic abnormality can be merely a condition of no great consequence to one individual but a genuinely severe problem to another.

In short, it seems clear that the major reason people seek orthodontic treatment is to minimize psychosocial problems related to their dental and facial appearance.[20] These problems are not "just cosmetic." They can have a major effect on the quality of life,[21] and the evidence presented in the final section of this chapter documents that orthodontic treatment can improve it.

Oral Function

Although severe malocclusion surely affects oral function, oral function adapts to form surprisingly well. It appears that malocclusion usually affects function not by making it impossible but by making it difficult, so that extra effort is required to compensate for the anatomic deformity. For instance, everyone uses as many chewing strokes as it takes to reduce a food bolus to a consistency that is satisfactory for swallowing, so if chewing is less efficient in the presence of malocclusion, either the affected individual uses more effort to chew or settles for less well-masticated food before swallowing it. Tongue and lip posture adapt to the position of the teeth so that swallowing rarely is affected (see Chapter 5). Similarly, almost everyone can move the jaw so that proper lip relationships exist for speech, so distorted speech is rarely noted even though an individual may have to make an extraordinary effort to produce normal speech. New data appears to indicate that Cl III malocclusions and anterior open bites severe enough to require orthognathic surgery can distort speech sounds and respond positively to surgical correction.[22,23] As methods to quantify functional adaptations of this type are developed, it is likely that more effects of malocclusion on speech function will be appreciated.

The relationship of malocclusion and adaptive function to TMD, manifesting with pain in and around the TMJ, is understood much better now than only a few years ago. The pain may result from pathologic changes within the joint but more often is caused by muscle fatigue and spasm. Muscle pain almost always correlates with a history of clenching or grinding the teeth as a response to stressful situations or of constantly posturing the mandible to an anterior or lateral position.

Some dentists have suggested that even minor imperfections in the occlusion serve to trigger clenching and grinding the teeth. If this were true, it would indicate a real need for perfecting the occlusion in everyone, to avoid the possibility of developing facial muscle pain. Because the number of people with at least moderate degrees of malocclusion (50% to 75% of the population) far exceeds the number with TMD (5% to 30%, depending on which symptoms are examined), it seems unlikely that dental occlusion alone is enough to cause hyperactivity of the oral musculature. A reaction to stress usually is involved. Some individuals react by clenching and grinding their teeth; others develop symptoms in other organ systems. An individual almost never has both ulcerative colitis (also a common stress-induced disease) and TMD.

Some types of malocclusion (especially posterior crossbite with a shift on closure, anterior open bite and Cl II and Cl III relationships) correlate positively with TMJ problems and other types do not, but even the strongest correlation coefficients are only 0.3 to 0.4. This means that for the great majority of patients, there is no association between malocclusion and TMD[24] or previous orthodontic treatment and TMD.[25] Therefore, orthodontics as the primary treatment for TMD almost never is indicated, but in special circumstances (see Chapter 18) it can be a useful adjunct to other treatment for the muscle pain.

Relationship to Injury and Dental Disease

Malocclusion, particularly protruding maxillary incisors, beyond 5 mm of overjet with incompetent lips can increase the likelihood of an injury to the teeth (Fig. 1.18).[26] About 15% to 18% of youth aged 12 to 18 experience permanent incisor trauma.[27] The majority of these children will incur only minor chips in the enamel.[28] For that reason, reducing the chance of injury when incisors protrude is not a strong argument for early treatment of all Class II problems (see Chapter 13), but with previous trauma and age younger than 9 years, the risk of additional trauma is 8.4 times higher than in children with no history of trauma.[29] For such a child, retracting the incisors (but not growth modification) is indicated. Extreme overbite, so that the lower incisors contact the palate, can cause significant tissue damage leading to early loss of the upper incisors and also can result in extreme wear of incisors. Both of these effects can be avoided by orthodontic treatment (see Chapters 11 and 12).

It is certainly possible that malocclusion could contribute to both dental decay and periodontal disease by making it

• **Fig. 1.18** Fractured maxillary central incisors in a 10-year-old girl. There is almost one chance in three of an injury to a protruding incisor, though fortunately the damage is rarely this severe. Most of the accidents occur during normal activity, not in sports.

harder to care for the teeth properly or by causing occlusal trauma. There is some weak evidence that malocclusion may be tied to periodontal health, but this may be dependant on overall oral health status and the association does not mean causation.[30] An individual's willingness and motivation determine oral hygiene much more than how well the teeth are aligned, and presence or absence of dental plaque is the major determinant of the health of both the hard and soft tissues of the mouth. If individuals with malocclusion are more prone to tooth decay, the effect is small compared with hygiene status. Occlusal trauma, once thought to be important in the development of periodontal disease, now is recognized to be a secondary, not a primary, etiologic factor. There is only a tenuous link between untreated malocclusion and major periodontal disease later in life.

Could orthodontic treatment itself be an etiologic agent for oral disease? Long-term studies have shown no indication that orthodontic treatment increased the chance of later periodontal problems.[31] The association between early orthodontic and later periodontal treatment appears to be only another manifestation of the phenomenon that one segment of the population seeks dental treatment while another avoids it. Those who have had one type of successful dental treatment, such as orthodontics in childhood, are more likely to seek another such as periodontal therapy in adult life.

In summary, it appears that both psychosocial and functional handicaps can produce significant need for orthodontic treatment. The evidence is less clear that orthodontic treatment reduces the development of later dental disease.

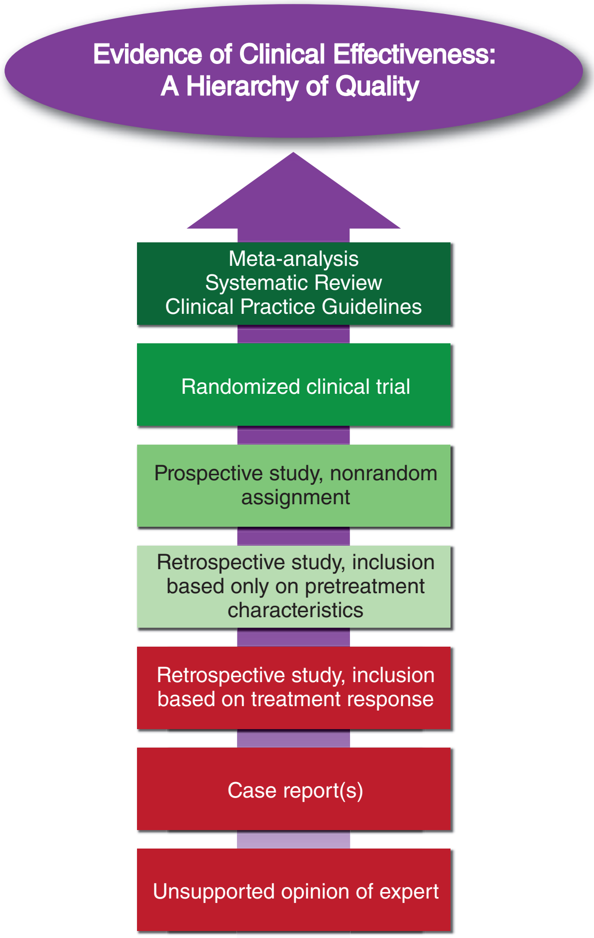
Generating and Assessing Data

Evidence-Based Selection

If treatment is needed, how do you decide what sort of treatment to use? The present trend in health care is strongly toward evidence-based treatment—that is, treatment procedures should be chosen on the basis of clear evidence that the selected method is the most successful approach to that particular patient's problem(s). The better the evidence, the easier the decision.

Meta-analysis, Systematic Reviews and Clinical Practice Guidelines: The Best Evidence

Orthodontics traditionally has been a specialty in which the opinions of leaders were important, to the point that



• **Fig. 1.19** Evidence of clinical effectiveness: a hierarchy of quality. Note that systematic reviews, by themselves, can have a wide range of quality.

professional groups coalesced around a strong leader. Angle, Begg, and Tweed societies still exist, and new ones whose primary purpose is to promulgate their leaders' opinions are still being formed in the 21st century. As any professional group comes of age, however, there must be a focus on evidence-based rather than opinion-based decisions. That very much includes orthodontics.

As Fig. 1.19 illustrates, a hierarchy of quality exists in the evidence available to guide clinical decisions. It reflects, more than anything else, the probability that an accurate conclusion can be drawn from the group of patients who have been studied. The unsupported opinion of an expert is the weakest form of clinical evidence. Often, the expert opinion is supported by a series of cases that were selected retrospectively from clinical practice records.

The problem with that, of course, is that the cases are likely to have been selected because they show the expected outcome. A clinician who becomes an advocate of a treatment method is naturally tempted to select illustrative cases that show the desired outcome, and if even they try to be objective, it is difficult to avoid introducing bias. When outcomes vary, as they often do, picking the cases that came out the way they were supposed to and discarding the ones that didn't is a great way to make your point (and usually invalid). Information based on selected cases, therefore, must be viewed with considerable reservation. One important way to control bias in reporting the outcomes of treatment is to be sure that *all* of the treated cases are included in the report.

For this reason, the gold standard for evaluating clinical procedures is the randomized clinical trial, in which patients are randomly assigned in advance to alternative treatment procedures. The great advantage of this method is that random assignment, if the sample is large enough (i.e., has enough power), should result in a similar distribution of all variables between (or among) the groups. Even variables that were not recognized in advance should be controlled by this type of patient assignment—and in clinical work, important variables often are identified only after the treatment has been started or even completed. Clinical trials in orthodontics are referred to throughout this book.

In randomized clinical trials, the studied individuals come from unbiased samples guided by inclusion and exclusion criteria and allocated on a blinded random basis. These studies are expensive and demanding to perform.

Systematic reviews are generated by studying a group of studies aimed at a similar purpose using criteria that evaluate the studies for all dimensions to ensure a reduced or low level of bias. These reviews can be even more effective when proper tools are used to evaluate the sample, methods and data (which have their own recommended methods), otherwise they can be harmful. The conclusions of the reviews draw conclusions, qualify the quality of the evidence, and point to future research directions.

If the group of studies is similar enough and uses comparable samples, methods, and reported outcomes, they can be statistically evaluated and weighted. Again, methods must be appropriate and tools must be available to evaluate these meta-analyses. This is an even higher level of analysis.

Orthodontic research is an excellent example of an area in which numerous small studies have been carried out toward similar ends, but differences in protocols and outcome variables were different enough to make comparisons difficult. That is why orthodontics should move to a core outcomes set that includes patient satisfaction so better comparisons can be made and we can approach bigger data sets. Meta-analyses are no substitute for new data collected with precise protocols, and including poorly done studies in a meta-analysis carries the risk of confusing rather than clarifying the issue.[32] Nevertheless, applying meta-analysis to clinical questions has considerable potential to reduce uncertainty about the best treatment methods.

An important caveat for meta-analyses is that the emphasis on statistical significance should not lead to overlooking the difference between statistical and clinical significance. Statistical significance evaluates the chance that a difference in the data set would be due just to the random variation that affects any group of treatment responses; clinical significance evaluates whether a difference of this magnitude would have any practical effect on the provision of treatment. Not all statistical differences are clinically significant, and sometimes differences that do not reach statistical significance nevertheless may indicate a clinical advance. That is why effect size or the quantification of differences between treatments introduced by Cohen is important.[33]

Unfortunately, randomized trials and meta-analysis cannot be used in many situations for ethical or practical reasons. For instance, a randomized trial of extraction versus nonextraction orthodontic treatment would encounter ethical concerns, would be very difficult and expensive to organize and manage if ethical difficulties could be overcome, and would require following patients for many years to evaluate long-term outcomes.

Another high quality level of evidence is the Clinical Practice Guideline. This is not an individual study, but a method to move from systemic reviews to the clinical setting, while evaluating the benefits and harms of the treatment options. This method includes expert panels and consensus development in an interdisciplinary manner. Again, recommendations are formulated using accepted tools.

Retrospective Studies: Control Group Required

Another acceptable way to replace opinion with evidence is by careful retrospective study of treatment outcomes under well-defined conditions. The best way to know—often the only way to know—whether a treatment method really works is to compare treated patients with an untreated control group. For such a comparison to be valid, the two groups must be equivalent before treatment starts. If the groups were not equivalent, they can be statistically adjusted, so you can say with confidence that posttreatment differences were due to the treatment.

There are difficulties in setting up control groups for orthodontic treatment. The principal ones are that the controls must be followed over a long period of time, equivalent to the treatment time, and that sequential radiographs usually are required. Radiation exposure for untreated children is problematic. At present, it is very difficult to get permission to expose children to x-rays that will be of no benefit to them personally. This means the longitudinal growth studies in the mid-20th century that used a series of cephalometric radiographs of untreated children cannot be repeated now. In the absence of newer data, they still are being used to provide control data in studies involving growth modification—although it is well established that in the United States and almost all other countries, children now grow larger and mature more quickly than at the time of those studies (see Fig. 3.7). When historic controls are the best that are available, it is better to have them than nothing, but the limitations must be kept in mind. Growth magnitudes and timing, along with so much else, have changed in the last 70 years.

A final important consideration is that what clinicians consider the important aspects of outcomes of treatment may or may not coincide with how patients perceive the outcome. In orthodontics, it is apparent that the appearance of the teeth on smile is a key outcome for patients. Fortunately, what the patients think now receives more attention than it did all the way through the 20th century, and data for the acceptable range of tooth display have become available recently.[15] Patient and practitioners often hold different viewpoints regarding the same dental feature and these perspectives must be weighed carefully. This has been demonstrated in many studies. Patient-centered treatment does not mean the patient is always right, but it does mean that the patient's point of view must be kept in mind both when treatment is planned and when its success is evaluated. But, practitioners cannot accept patient-generated treatment plans that do not meet the standard of care.

The era of orthodontics as an opinion-driven specialty is clearly at an end. In the future, it will be evidence informed, which is all for the best. This means evidence that is applied to maximize the results for *this* patient given their esthetic, functional, physical, behavioral, and financial preferences and limitations AND given the ability and experience of the practitioner. Limited care for an adolescent may be just as appropriate as comprehensive care for an adult and vice versa given these constraints.

In the meantime, clinical decisions must still be made using the best information currently available. When the latest new method appears with someone's strong recommendation and a series of case reports in which it worked very well, it is wise to remember the aphorism "Enthusiastic reports tend to lack controls; well-controlled reports tend to lack enthusiasm."

In this and the subsequent chapters, recommendations for treatment are based insofar as possible on solid clinical evidence. When this is not available, the authors' current opinions are provided and labeled as such.

Artificial Intelligence in Orthodontics

At present, the application of artificial intelligence (AI) in orthodontics is still in its infancy. However, AI applications to digital orthodontics is obvious, and there are increasing AI tools available for practitioners. It is important to understand AI terminologies (machine learning versus deep learning), principles (algorithms such as convolutional neuron network), and clinical applications. Digital dentistry is important because it allows AI to impact a traditional practice.

Traditional orthodontic workflow. Orthodontists need to perform many tasks while focusing on important information that will lead to the correct decisions and actions. These skills are critical for treatment success. Many are repetitive and include taking patient records, analyzing the records, summarizing diagnoses and treatment plans, placing appliances, adjusting orthodontic mechanics and forces, finishing and retention. In a conventional practice, clinicians execute these tasks themselves by chairside observation, writing a treatment plan and then performing treatment like bending wires, and so on.

Digital orthodontics. Recent digital technology has revolutionized these tasks. Hardware such as digital cephalometric and panoramic radiography, cone beam computer tomography (CBCT), intra-oral 3D scanners and printers have helped in gathering digital data. Software has been useful in representing these data like digital electronic records, graphic tracings, and 3D imaging renditions for analysis.

Digital workflow. Transition from traditional workflow to digital dentistry requires a change in clinical setup and workflow, which involves the use of advanced technologies and software to plan, design and execute orthodontic treatment. Digital radiographs allow for faster image acquisition, reduced radiation exposure, and the ability to store and transmit images electronically with the immediate reconstruction of digital images, on a computer screen. The use of 3D CBCT can render 3D images, which adds information that helps locate, plan, and treat anomalies within tissues like impacted teeth, supernumerary teeth, craniofacial anomalies and facial asymmetries.

Optical scanning technology has become increasingly prevalent in acquiring three-dimensional information for contemporary orthodontics. The 3dMDface System (3dMD LLC, Atlanta, US) allows acquisition of 3D surface topology and the color texture of a patient's face. The 3D scan provides higher resolutions for quantitative assessment of facial expression and animation. Researchers have applied the method to study surgical outcomes of reconstruction of patients with congenital orofacial anomalies.

The intra-oral scanner is the most popular technology, which replaces conventional impressions. Many orthodontic offices have already adopted intra-oral scan as a routine procedure so the images can be transmitted and printed quickly and accurately.

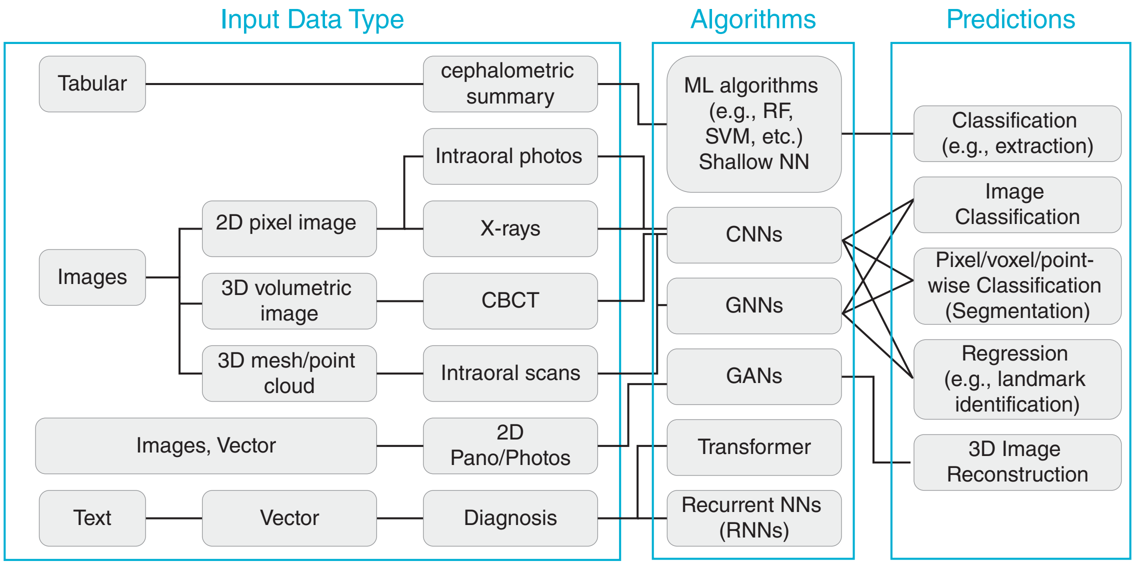
Orthodontic diagnosis and treatment planning. Operations of traditional practice and contemporary digital orthodontics share the same purpose, which is to provide optimal diagnosis and treatment planning for patient care. Since the 1920s orthodontists have accumulated data to describe malocclusion, faces, and growth patterns. From the data, problem lists and treatment plans were generated. As large data bases were accumulated, bioinformatics applications began assisting clinicians in making medical diagnoses and predicting outcomes. In orthodontics, diagnosis and treatment planning of every patient includes data classification, imaging analysis, treatment, and monitoring progress. Because there are 5 million people undergoing orthodontic treatment in the United States per year, these processes are repeated 5 million times annually. This is a repetitive process and artificial Intelligence (AI) is particularly useful for dealing with repetitive and tedious jobs.

Artificial intelligence (AI). Today, AI can suggest options for decisions, prioritization, and planning. There are two types of AI, commonly defined as weak and strong AI. Weak AI, also known as narrow AI, refers to artificial intelligence designed to perform a specific task or set of tasks.[34,35] These tasks include identifying objects in images. However, it does not possess the broad, adaptable intelligence associated with human cognition. Most current orthodontic AI applications (classification, regression, image segmentation, and landmark identification), are weak AI.

On the other hand, strong AI, also known as artificial general intelligence (AGI), refers to AI that exhibits human-like cognitive abilities and can learn and reason in a manner that is indistinguishable from human intelligence.[36,37] A sophisticated multimodel AI for diagnosis and treatment planning could be classified in this category.

AI applications. Specific neural network algorithms should be selected according to the types of input data and outcome predictions (Fig. 1.20). Neural networks can be applied to a wide range of data types and tasks, making them a powerful tool for artificial intelligence and machine learning. Those data types include tabular data, images, and text. Although neural networks are flexible for many data types, it is important to use a suitable neural network in order to effectively and efficiently learn underlying patterns from the corresponding data. In this section, we will discuss various AI algorithms and their related data types for different clinical applications.

Supervised machine learning. A major subset of AI algorithms is "Machine Learning" (ML), which focuses on the development of algorithms and statistical models that allow computer systems to learn and make predictions or decisions without being explicitly programmed. ML algorithms can be broadly categorized into supervised learning, unsupervised learning, and reinforcement learning. Tabulated data with predetermined features (e.g., orthodontic crowding, overjet, overbite, and cephalometric measures) are most suitable for training machine learning. For example, an important part of orthodontic treatment is to determine whether to extract or not to extract permanent teeth. Researchers have used supervised machine learning algorithms to study extraction decisions. The predictability ranges from 80% to 95%, depending



• **Fig. 1.20** Neural networks can be applied to a wide range of data types and tasks, but they must be used with the appropriate data types to be productive and obtain the required outcomes. *CBCT*, Cone Beam Computer Tomography; *ML*, Machine Learning; *RF*, Radio Frequency; *SVM*, Support Vector Machine; *NN*, Neural Network; *CNN*, Convolutional Neural Network; *GNN*, Graph Neural Network; *GAN*, Generative Adversarial Network; *RNN*, Recurrent Neural Network.

upon the method of sample collections and the type and amount of information provided. A critical factor is what is really the right answer or "ground truth." Is the ground truth one practitioner's opinion or the opinion of multiple expert practitioners or a true gold standard? A method with diverse providers would require bigger data for the machine to learn. It is possible that an AI model, trained from a diverse population, could predict extractions across different practice settings.

Unsupervised machine learning. The examples of unsupervised machine learning (no predetermined ground truth) are clusterings of shapes such as the shape of the mandibular condyle or a pattern classification of craniofacial form. Shape analysis is the automatic analysis of geometric shapes. When analyzing ten unilateral cleft palatal defects, the first three geometric descriptors (principal components) are: volume, curvature, and transverse dimension of the cleft (Fig. 1.21.) A complete shape descriptor is a representation that can be used to completely reconstruct the defective object.

Deep learning. Deep learning is a subset of machine learning that focuses on training artificial neural networks with multiple layers to learn and extract complex patterns and representations from data. It is inspired by the structure and function of the human brain, particularly the interconnected network of neurons. Traditional machine learning algorithms often require manual adjustments, where humans handcraft relevant features from the data. In deep learning, however, neural networks are designed to automatically learn hierarchical representations of data through multiple layers of interconnected nodes, called neurons or units. Each layer of neurons performs transformations on the input data, gradually extracting higher-level features and representations.

Deep learning has the potential, if managed carefully, to make an impact on orthodontics. This is possible due to the ability of deep neural networks to automatically learn and discover intricate patterns and representations directly from raw data, eliminating the need for manual adjustments. Artificial neural networks (ANNs) are a core element of deep learning algorithms. Recurrent neural network (RNN) and convolutional neural network (CNN) are the most common network architectures in deep learning. The most obvious practical examples are auto-landmarking of cephalometric images and analysis, automatic analysis of panoramic X-rays, auto-segmentations of CBCT images and intra-oral scan surfaces.

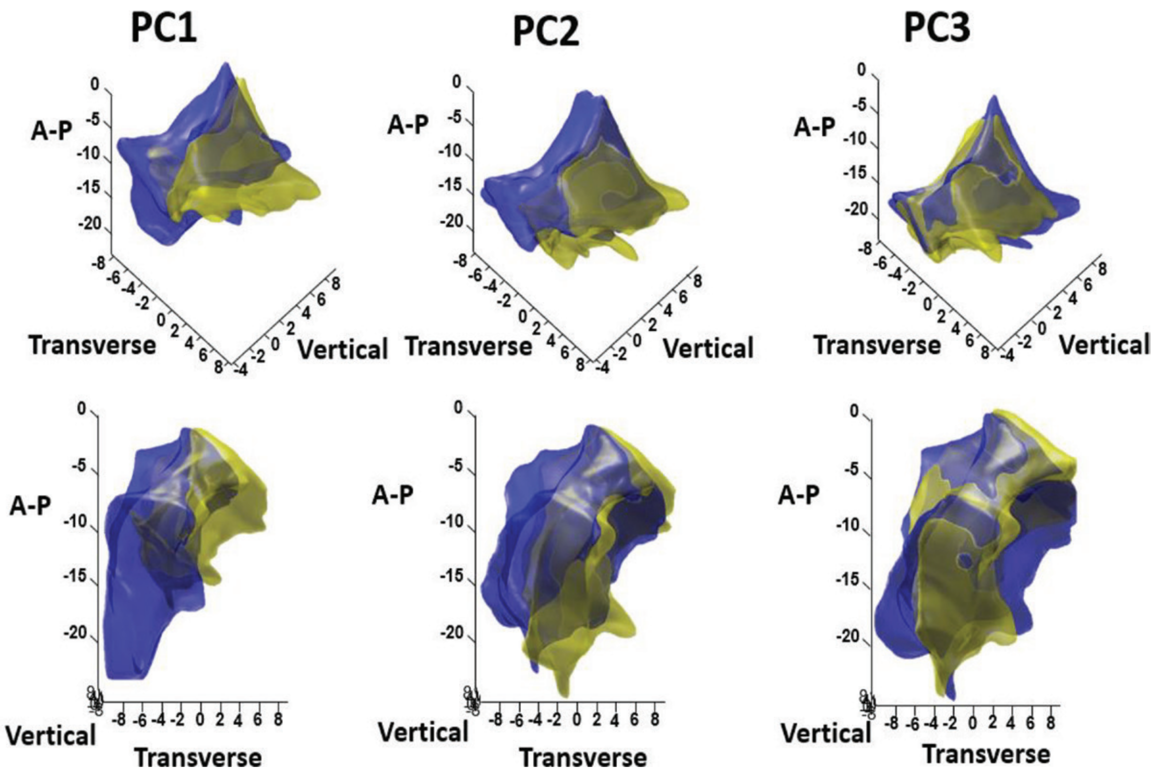
Convolutional neural network (CNN). A deep network AI model was reported to automatically detect cephalometric landmarks based on X-ray cephalograms from various manufacturers and CBCT-generated cephalograms.[38] The mean radial error of the average value of all landmarks was 1.42 mm. This type of software could streamline orthodontic data gathering and analysis (Phimentum [Newton, MA] and CephPro3D Largev [Beijing, China]).

There is an industrial software that can automatically label tooth numbers and perform pathological and non-pathological detection, including impacted teeth, caries, third molars, periapical anomalies, missing teeth, supernumerary teeth, retained deciduous teeth, residual root, residual crown, alveolar bone resorption, low/high-density jaw anomalies, dental implant, crown, filling, and root canal treatment (Fig. 1.22). This could allow orthodontists to formulate diagnostic conclusions supplemented by an AI assistant's second opinion, significantly reducing the risk of missed diagnoses.

CBCTs consist of voxels in a sequential order. CNN can be used to automatically segment a CBCT into the foreground teeth and background craniofacial bones. Sheng and coworkers derived a sophisticated CNN algorithm from 4125 annotated CBCTs. They showed that the AI model can precisely auto-segment orofacial bone and teeth. Some commercial software can automatically segment and reconstruct the teeth, maxilla, mandible, maxillary sinus, inferior alveolar canal, airway, and soft tissue profiles (Fig. 1.23).

Applying similar algorithms to a canine impaction case, Fig. 1.24 shows where the canine is trapped between the roots of the central and lateral incisors. The original (unsegmented) CBCT shows a blurred image of the impacted canine. The auto-segmentation offers clinicians the information to guide canine eruption with minimum damage to the roots of incisors.

Another example of CNN applications is to automatically determine dental crowding using clinical intraoral photos. Using



• **Fig. 1.21** Principal components (PC) of shape analysis of the defect in the unilateral palatal cleft. PC1: Volume of the cleft; PC2: Curving of the cleft; PC3: Transverse of the cleft.

835 patients and maxillary occlusal photos, residual neural network (Resnet) returned 84% predicted crowding with an error ≤3 mm. Although the Resnet model applying CNN to measure crowding of images is promising, future research will use standardized photos and the exclusion of confounding areas of the image to reduce prediction errors. The previous work suggests CNN's capability of learning orthodontic features, which may improve workflow efficiency in orthodontic treatment and education.

Graphic neural network (GNN). 3D surface data from the intra-oral scanner consists of a discrete set of points in space. These data, do not possess a sequential order nor do the neighboring points have a uniform relationship. This format is more suitable for GNN, which deals with geographic features. Many companies (e.g., Align Tech, ULab System, 3M-UNITEK, Henry Schein) have their own AI program for tooth segmentation. They can use this technology to manufacture products such as aligners. In addition, automated landmark identification of individual teeth from the intra-oral scan has allowed defining tooth orientation and anatomic features.[38,39] Researchers have utilized these technologies to quantify tooth movement from clinical data sets and compare outcomes between different orthodontic modalities. Murphy et al.[40] found that clear aligner achieved smaller movements than that of the conventional bracket-wire system. These evidence-based studies using big data are becoming available with the use of these AI tools.

It appears the future of AI in orthodontics, when supervised, can be a productive clinical tool in terms of options for diagnosis and treatment planning. It can also provide new information regarding treatment methods not previously available as long as the ground truth is available for development and testing.

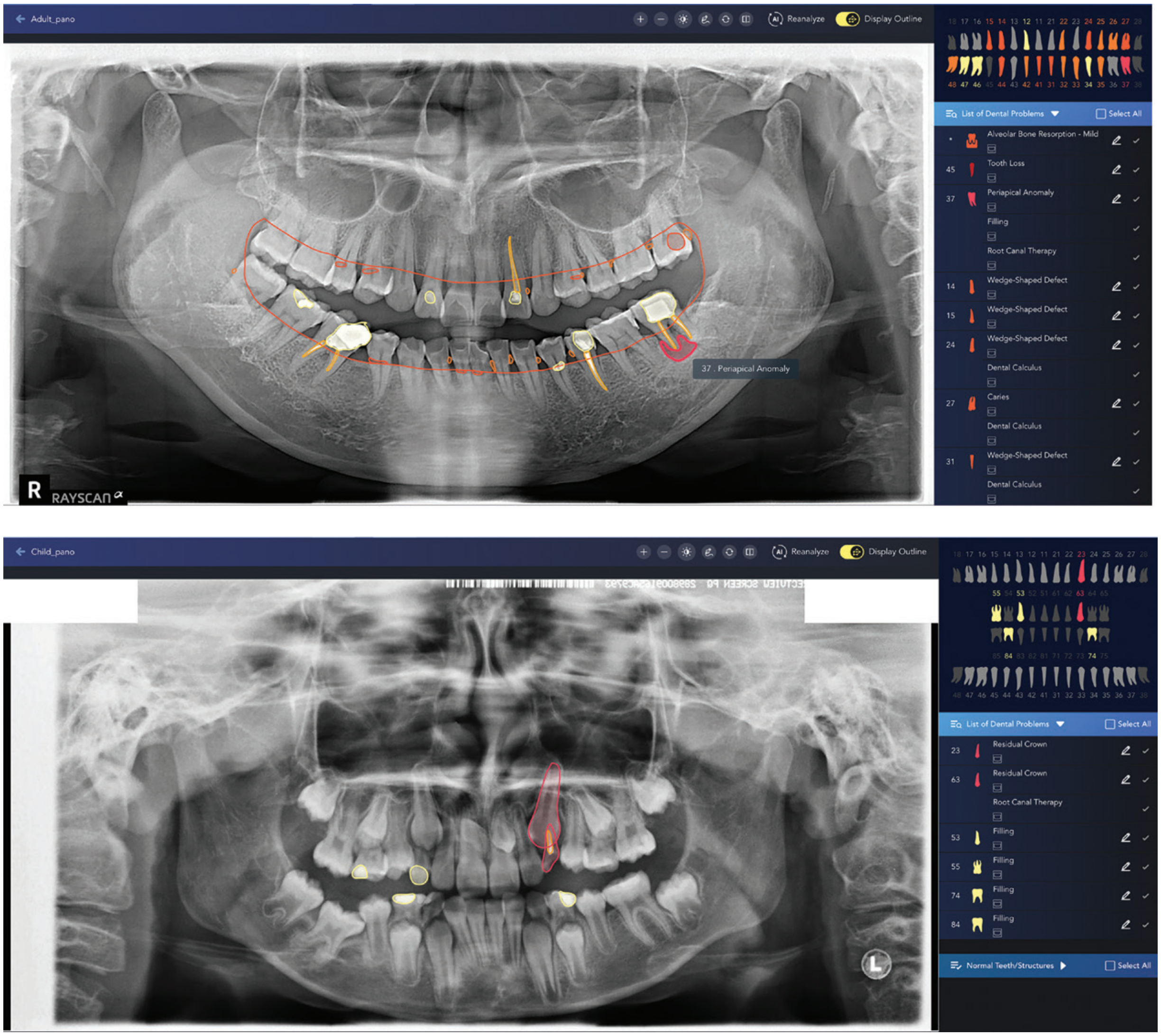
Demand for Treatment

Epidemiologic Estimates of Orthodontic Treatment Need

Psychosocial and facial considerations, not just the way the teeth fit, play a role in defining orthodontic treatment need. For this reason, it is difficult to determine who needs treatment and who does not just from an examination of dental casts or radiographs. Nevertheless, it seems reasonable that the severity of a malocclusion correlates with need for treatment, and as we will discuss in more detail here, there is good evidence to support that correlation. This assumption is necessary when treatment need is estimated for population groups.

Several indices for scoring how much the teeth deviate from the normal, as indicators of orthodontic treatment need, were proposed in the 1970s but not widely accepted for the screening of potential patients. There are now two major methods for scoring the severity of malocclusion: the peer assessment rating (PAR) system, developed in the United Kingdom, and the American Board of Orthodontics (ABO) discrepancy index, developed in the United States. It is important to keep in mind that these systems consider just the dentition, not skeletal or facial characteristics.

PAR scores are calculated from measurements of maxillary and mandibular anterior alignment (crowding and spacing), buccal segment occlusion (anteroposterior, transverse, and vertical), overjet or reverse overjet, overbite, and midline discrepancies, with use of a weighting scale for each characteristic.[41] ABO index scores are calculated similarly, with the difference primarily that it adds three cephalometric measurements.[42] The PAR analysis is valuable as a



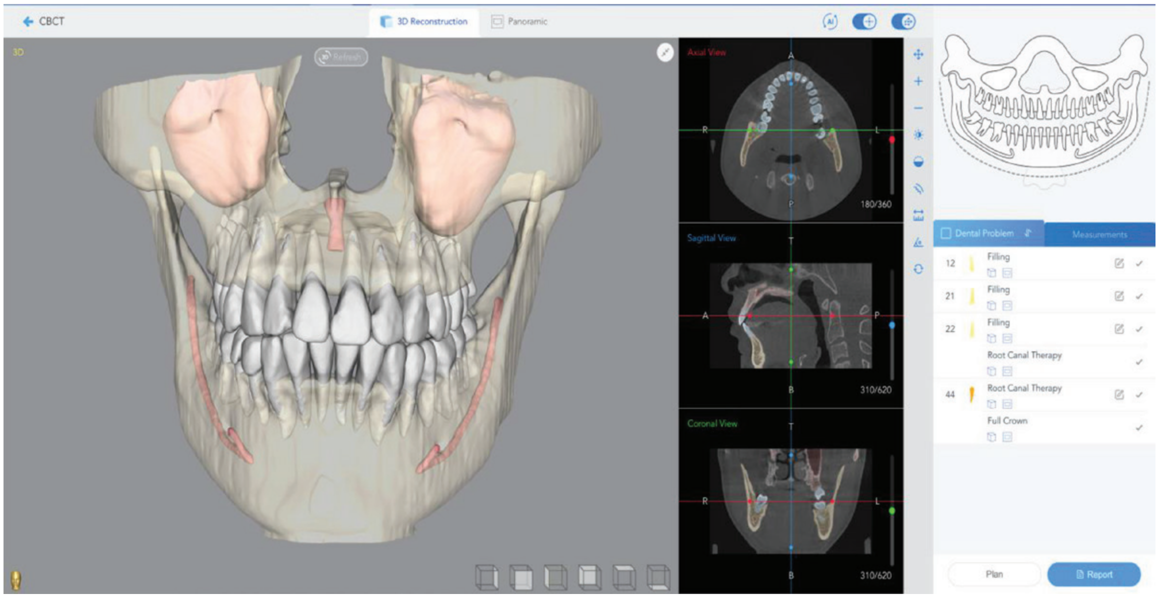
• **Fig. 1.22** AI programs can identify pathologic and nonpathologic elements of images to serve as a second opinion for examination data. (Courtesy of DeepCare Inc.)

pre- and posttreatment tool to measure both conditions and the improvement. The ABO index evaluates the final result of treatment against a standard. Both systems have been shown to correlate reasonably well with expert opinions of orthodontic treatment need.

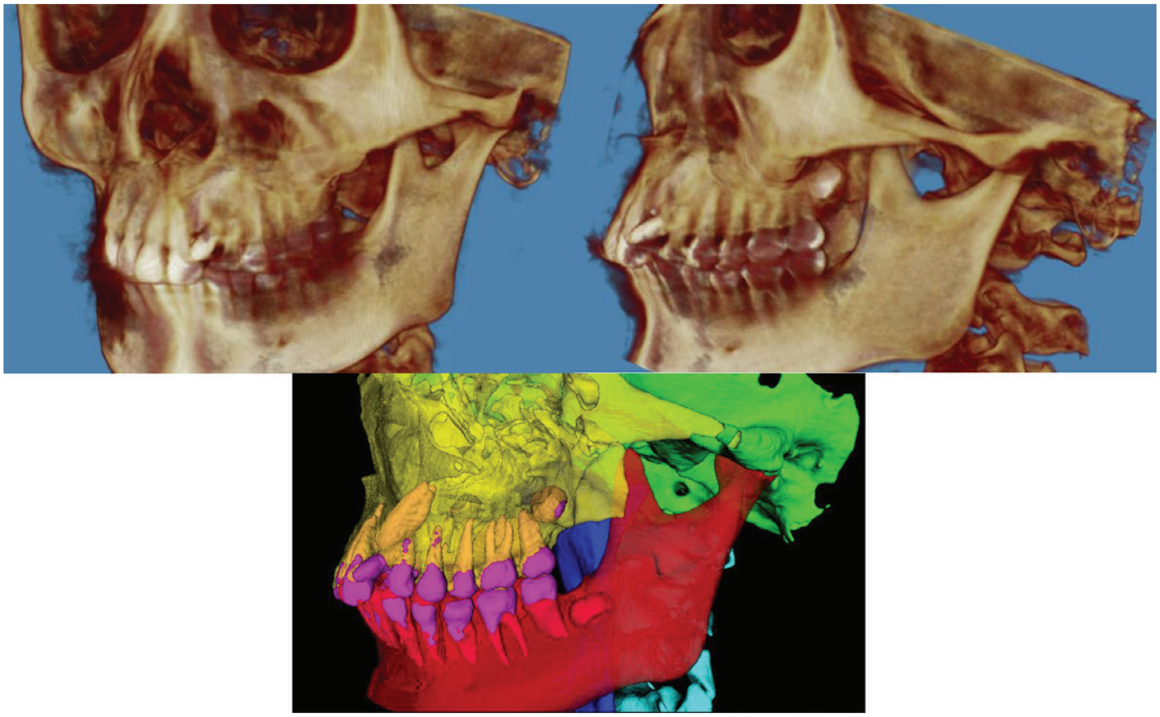
The Index of Treatment Need (IOTN), developed by Brook and Shaw in the United Kingdom,[43] was designed to evaluate need for treatment. It places patients in five grades from "no need for treatment" to "treatment required" that correlate reasonably well with clinician's judgments of need for treatment. The index has a dental health component derived from occlusion and alignment (Box 1.1 outlines the criteria and an esthetic component derived from comparison of the patient's dental appearance versus standard photographs (Fig. 1.25). An AI method trained and tested on novel images demonstrated sensitivity = 0.77, specificity = 0.88, PPV = 0.89 and NPV = 0.75 assigning the esthetic component as 1–5 or 6–10, essentially no treatment need vs treatment need.[44] There is a surprisingly good correlation between treatment need assessed by the dental health and esthetic components of IOTN.[45]

With some allowances for the effect of missing teeth, it is possible to calculate the percentages of US children and youths who would fall into the various IOTN grades from the NHANES III data set.[46] Fig. 1.26 shows the percentage of youths age 12 to 17 in the three major racial or ethnic groups in the US population estimated with IOTN to have mild, moderate, or severe treatment need and the percentage who had treatment at that time. As the graph shows, the number of White youths who received treatment was considerably higher than the number of Black or Mexican American children ($P < 0.001$). Treatment almost always produces an improvement but may not totally eliminate all the characteristics of malocclusion, so the effect is to move some individuals from the severe to the mild treatment need categories. The higher proportion of severe malocclusion among Blacks probably reflects more treatment in the White group, which moved them down the severity scale, rather than the presence of more severe malocclusion in the Black population.

How do the IOTN scores compare with what parents and dentists think relative to orthodontic treatment need? The existing (rather weak) data suggest that in typical American neighborhoods, about 35% of adolescents are perceived by parents and peers as needing orthodontic treatment. Note that this is larger than the number of children who would be placed in IOTN grades 4 and 5 as having severe problems and definitely needing



• **Fig. 1.23** By segmenting the teeth and other structures, like the maxilla, mandible, sinus and some nerves from the background, structures can be more apparent for evaluation. (Courtesy of DeepCare Inc.)



• **Fig.1.24** Here is a contrast of two images. The upper image is from a standard CBCT and the lower segmented image that provides an enhanced view of the impacted maxillary left canine. This makes determining the status of the tooth and surrounding teeth as well as potential methods and direction of traction more discernable.

treatment, but smaller than the total of grades 3, 4, and 5 for moderate and severe problems.

Dentists usually judge that only about one-third of their patients have normal occlusion, and they suggest treatment for about 55% (thereby putting about 10% in a category of malocclusion with little need for treatment). It appears that they include all the children in IOTN grade 3 and some of those in grade 2 in the group who would benefit from orthodontics. Presumably, facial appearance and psychosocial considerations are used in addition to dental characteristics when parents judge treatment need or dentists decide to recommend treatment.

Who Seeks Treatment?

Demand for treatment is indicated by the number of patients who actually make appointments and seek care. Not all patients with malocclusion, even those with extreme deviations from the norm, seek orthodontic treatment. Some do not recognize that they have

• BOX 1.1 Index of Treatment Needs (IOTN) Treatment Grades

Grade 5 (Extreme/Need Treatment)

5.i Impeded eruption of teeth (except third molars) due to crowding, displacement, the presence of supernumerary teeth, retained deciduous teeth, and any pathologic cause.
5.h Extensive hypodontia with restorative implications (more than one tooth per quadrant) requiring preprosthetic orthodontics.
5.a Increased overjet greater than 9 mm.
5.m Reverse overjet greater than 3.5 mm with reported masticatory and speech difficulties.
5.p Defects of cleft lip and palate and other craniofacial anomalies.
5.s Submerged deciduous teeth.

Grade 4 (Severe/Need Treatment)

4.h Less extensive hypodontia requiring prerestorative orthodontics or orthodontic space closure (one tooth per quadrant).
4.a Increased overjet greater than 6 mm but less than or equal to 9 mm.
4.b Reverse overjet greater than 3.5 mm with no masticatory or speech difficulties.
4.m Reverse overjet greater than 1 mm but less than 3.5 mm with recorded masticatory or speech difficulties.
4.c Anterior or posterior crossbites with greater than 2 mm discrepancy between retruded contact position and intercuspal position.
4.l Posterior lingual crossbite with no functional occlusal contact in one or both buccal segments.
4.d Severe contact point displacements greater than 4 mm.
4.e Extreme lateral or anterior open bites greater than 4 mm.
4.f Increased and complete overbite with gingival or palatal trauma.
4.t Partially erupted teeth, tipped, and impacted against adjacent teeth.
4.x Presence of supernumerary teeth.

Grade 3 (Moderate/Borderline Need)

3.a Increased overjet greater than 3.5 mm but less than or equal to 6 mm with incompetent lips.
3.b Reverse overjet greater than 1 mm but less than or equal to 3.5 mm.
3.c Anterior or posterior crossbites with greater than 1 mm but less than or equal to 2 mm discrepancy between retruded contact position and intercuspal position.
3.d Contact point displacements greater than 2 mm but less than or equal to 4 mm.
3.e Lateral or anterior open bite greater than 2 mm but less than or equal to 4 mm.
3.f Deep overbite complete on gingival or palatal tissues but no trauma.

Grade 2 (Mild/Little Need)

2.a Increased overjet greater than 3.5 mm but less than or equal to 6 mm with competent lips.
2.b Reverse overjet greater than 0 mm but less than or equal to 1 mm.
2.c Anterior or posterior crossbite with less than or equal to 1 mm discrepancy between retruded contact position and intercuspal position.
2.d Contact point displacements greater than 1 mm but less than or equal to 2 mm.
2.e Anterior or posterior open bite greater than 1 mm but less than or equal to 2 mm.
2.f Increased overbite greater than or equal to 3.5 mm without gingival contact.
2.g Prenormal or postnormal occlusions with no other anomalies.

Grade 1 (No Need)

1. Extremely minor malocclusions, including contact point displacements less than 1 mm.

a problem; others feel that they need treatment but cannot afford it or cannot obtain it.

Both the perceived need and demand vary with social and cultural conditions. More children in urban areas are thought (by parents and peers) to need treatment than children in rural areas. Family income is a major determinant of how many children receive treatment (Fig. 1.27). This appears to reflect two things: not only that higher income families can more easily afford orthodontic treatment, but also that good facial appearance and straight teeth are associated with more prestigious social positions and occupations. The higher the aspirations for a child, the more likely the parents are to seek orthodontic treatment for him or her.

Why do they seek treatment for their children? We have already noted that psychosocial handicaps are the major reason. Another way to put this issue is "Does having a less than ideal smile affect the way people act and live?" This question was examined by the American Dental Association's Health Policy Institute in 2015.[47] An online survey was conducted by the Harris Poll, and nearly 15,000 responses from a randomly selected group of individuals age 18 and older were analyzed. The study group was evaluated as a whole, by economic status (low, middle, and high household income), and by age (18–34, 35–49, 50–64, and 65 or older). This national data set tells an interesting story related to dental esthetics. Twenty-nine percent of low-income adults and 28% of young adults (18–34) believed the appearance of their mouth and teeth affected their ability to interview for a job. That is over one-fourth of these groups. Twenty-five percent of all adults said they avoid smiling, 23% feel embarrassed, and 20% experience anxiety because of the condition of their mouth and teeth. But low-income and young adults felt the greatest impact, with a minimum of 30% in each of these two groups indicating that they experienced a problem related to the appearance of their teeth very often or occasionally. Finally, 82% of all responders agreed with the statement "It is easier to get ahead in life if I have straight, bright teeth."

So, although the need for treatment and its assessments and benefits are usually determined with carefully quantified dental morphologic and degrees of craniofacial deformity, poor dental esthetics is enough to clearly impair people. Often, we lose track of that simple truth by trying to justify orthodontic treatment at a higher and seemingly more significant level. In fact, people value straight teeth because it makes their lives easier and better.

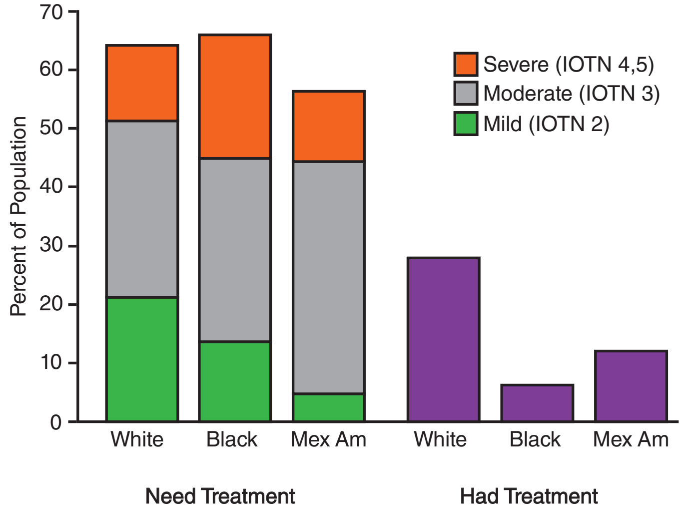
Because it is widely recognized now that severe malocclusion can affect an individual's entire life, every US state now provides at least some orthodontic treatment for low-income families through its Medicaid program. Nevertheless, Medicaid and related programs support only a tiny fraction of the population's orthodontic care. From that perspective, it is interesting that even in the lowest income group, almost 5% of youths and over 5% of adults report having received treatment; 10% to 15% at intermediate income levels have received treatment. This indicates the importance placed on orthodontic treatment by families who judge that it is a factor in social and career progress for their children.

The effect of financial constraints on demand can be seen most clearly by the response to third-party payment plans. When third-party copayment is available, the number of individuals seeking orthodontic treatment rises considerably, but even when all costs are covered, some individuals for whom treatment is recommended do not accept it. It seems likely that under optimal economic conditions, demand for orthodontic treatment will at least reach the 35% level thought by the public to need treatment.

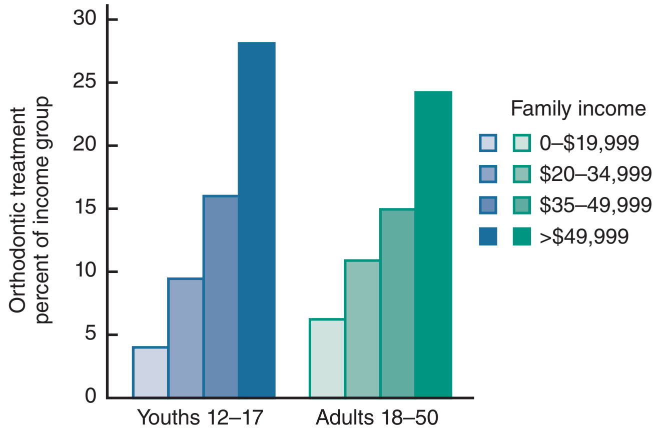
• **Fig. 1.25** The representative standard photographs of the Index of Treatment Need (IOTN) esthetic index. The score is derived from matching the esthetics of the malocclusion being observed to the appropriate standard photograph. Number 1 is the most attractive and number 10 the least attractive arrangement. Grades 8–10 indicate definite need for orthodontic treatment; 5–7, moderate or borderline need; 1–4, no or slight need.

In higher socioeconomic areas in the United States, 35% to more than 50% of children and youths now are receiving orthodontic care. In Switzerland, where high average incomes and supplemental social programs mean that essentially all citizens who want treatment can get it, 56% of the 2012 population aged 15 to 24 years were receiving or had received orthodontic treatment.[48] Acceptance of treatment is at similar levels in the Scandinavian countries for the same reasons.

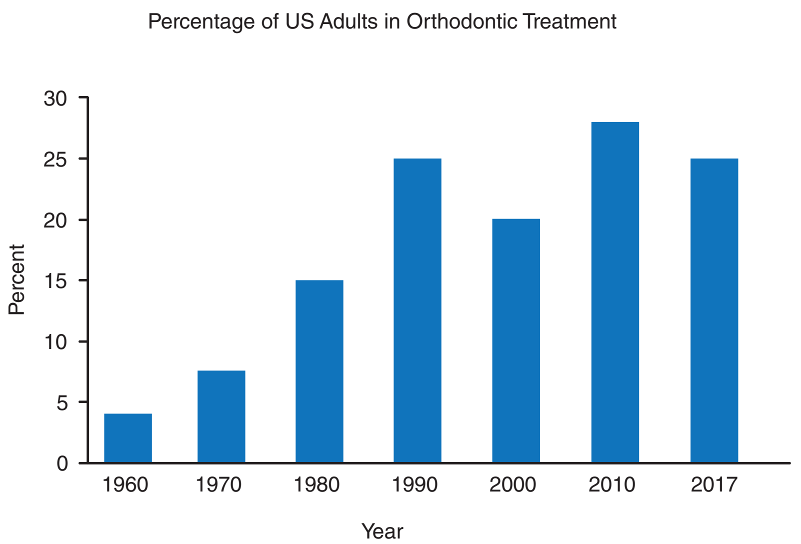
Orthodontic treatment for adults was rare until the latter half of the 20th century. In the 1960s, only 5% of all orthodontic patients in the United States were adults (age 19 or older). By 1990, about 25% of all orthodontic patients were adults (18 or older) (Fig. 1.28). It is interesting to note that the absolute number of adults seeking orthodontic treatment remained constant for the next decade while the number of younger patients grew, so by 2000 the proportion of adults in the orthodontic patient population had dropped to about 20%. By 2010 it had increased again to over 25% of the total, and the most recent survey by the American Association of Orthodontists (2017) indicated a little over 25% were being treated.



• **Fig. 1.26** Orthodontic need by severity of the problem for White, Black and Mexican American youths age 12–17 in the United States, 1989–94, and the percentage of each group who reported receiving previous orthodontic treatment. Approximately 12–20% have severe malocclusions. The greater number of Whites who received treatment probably accounts for the greater proportion of mild and moderate problems in the White population.



• **Fig. 1.27** The percentage of the US population, 1989–94, who received orthodontic treatment, as a function of family income. Although severe malocclusion is recognized as an important problem and all states offer at least some coverage to low-income children through their Medicaid programs, this funds treatment for a very small percentage of the population. Nevertheless, nearly 5% of the lowest income group and 10%–15% of intermediate income groups reported some orthodontic treatment. This reflects the importance given to orthodontic treatment—it is sought even when it stretches financial resources in less-affluent families.



• **Fig. 1.28** From the mid-20th century, when almost no adults received orthodontic treatment, to the 1990s, there was an almost steady rise in the number of adult patients. In the 1980s, a "baby bust" period, the increasing number of adult patients was the major source of the overall increase in orthodontics, whereas in the 1990s, a "baby boom" period, the number of adult patients increased a little but most of the growth involved treatment of children, so the adult percentage declined. There was a further increase in the number of adults in treatment and their percentage of the total patient population in the first decade of the 21st century, bringing the percentage back to 25%–30%.

Many adult patients indicate that they wanted treatment earlier but did not receive it, often because their families could not afford it; now they can. Wearing braces as an adult is more socially acceptable than it was previously. Clear aligners have made orthodontic treatment even more acceptable to many patients. Recently, an increased number of older adults (40 and over) have sought orthodontics, usually in conjunction with other treatment, to save their teeth, and the majority of that oldest subgroup were male (every other age group from childhood on has more females). As the population ages, these older adults are likely to be the fastest growing group who seek orthodontic treatment.

Many of the children and adults who seek orthodontic treatment today have dentofacial conditions that are within the normal range of variation, at least by definitions that focus tightly on obvious degrees of handicap. Does that mean treatment is not indicated for those with lesser problems? Today, medical and dental interventions that are intended to make the individual either "better than well" or "beyond normal" are called *enhancements*.

Typical medical and surgical enhancements are drugs to treat erectile dysfunction, face lifts, and hair transplants. In dentistry, a good example of enhancement is tooth bleaching.

In this context, orthodontics often can be considered an enhancement technology. It is increasingly accepted that appropriate care for individuals should often include enhancement to maximize their quality of life. If you really want it because you are convinced you need it, perhaps you really do need it—whether it is orthodontics or many other types of treatment. Medicaid and Medicare and many insurance companies now have accepted the reality that at least some enhancement procedures have to be accepted as reimbursable medical expenses. Similarly, when orthodontic benefits are included in insurance coverage, the need for treatment is no longer judged just by the severity of the malocclusion. The bottom line: Enhancement is appropriate dental and orthodontic treatment, just as it is in other contexts.

A key question, of course, is "Does orthodontic treatment really increase quality of life and self-esteem?" A number of studies have documented improvement in quality-of-life scores and self-esteem in children and adolescents,[49] and reports have shown quality-of-life effects after orthodontic treatment in children of African, European, and Asian descent.[50–52] Multiple studies have shown that this is true for adults as well, and the range of improvements in quality of life extend further than one might have thought. For instance, a Brazilian study showed that adults with ideal smiles are considered to be more intelligent and have a greater chance of finding a job,[53] and a systematic review documented patient satisfaction after orthodontic treatment combined with orthognathic surgery.[54] The data can be summarized succinctly: If your dental and facial appearance differs significantly from that of your group, you benefit socially from correcting it.

Orthodontics has become a more prominent part of dentistry in recent years, and this trend is likely to continue. The vast majority of individuals who had orthodontic treatment feel that they benefited from the treatment and are pleased with the result. Not all patients have dramatic changes in dental and facial appearance, but nearly all recognize an improvement in both dental condition and psychologic well-being.

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2

Concepts of Growth and Development

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A thorough background in craniofacial growth and development is necessary for every dentist. Even for those who never work with children, it is difficult to comprehend conditions observed in adults without understanding the developmental processes that produced these problems. For those who interact professionally with children—and almost every dentist does so at least occasionally—it is important to distinguish normal variation from the effects of abnormal or pathologic processes. Because dentists and orthodontists are heavily involved in the development of not just the dentition but the entire dentofacial complex, a conscientious practitioner may be able to manipulate facial growth for the benefit of the patient. It is not possible to do so without a thorough understanding of both the pattern of normal growth and the mechanisms that underlie it.

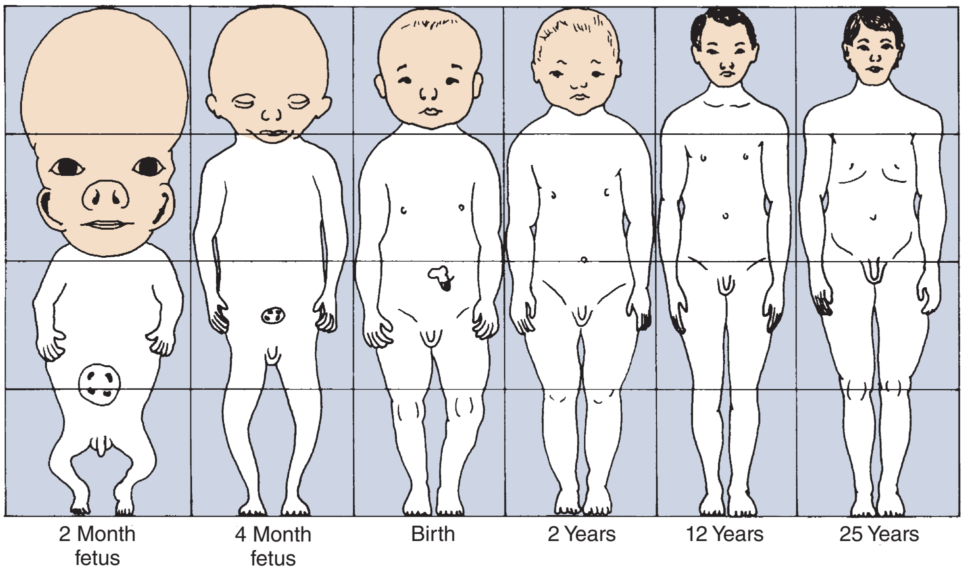
The very terms *growth* and *development* can cause difficulties in understanding. Growth and development, although closely related, are not synonymous. In conversational English, *growth* usually refers to an increase in size but tends to be linked more to change than anything else. Only if growth meant change, after all, could someone seriously speak of a period of economic recession as one of "negative economic growth." Some tissues grow rapidly and then shrink or disappear, so a plot of physical growth versus time may include a negative phase. On the other hand, if growth is defined solely as a process of change, the term becomes almost meaningless. In this chapter, the term growth usually refers to an increase in size or number.

As a general term, *development* connotes an increasing degree of organization, often with unfortunate consequences for the natural environment. With reference to growth, the term *development* is used almost always to refer to an increase in complexity, and it is used in that way in this chapter. Development carries an overtone of increasing specialization, so that one price of increased development is a loss of potential. Growth is largely an anatomic phenomenon, whereas development is physiologic and behavioral.

Although dentists work with the physical features of the teeth and face, a major reason for orthodontic treatment is its psychosocial effects. Furthermore, patient cooperation is necessary, and eliciting it in children of different ages requires a knowledge of social and behavioral development. Both physiologic and psychosocial development are important subjects for this chapter. For convenience, not because they are innately more important, physical growth concepts are presented first, and then behavioral developmental factors are reviewed.

Growth: Pattern, Variability, and Timing

In studies of growth and development, the concept of pattern is important. In a general sense, pattern (as in the pattern from which articles of clothing of different sizes are cut) reflects

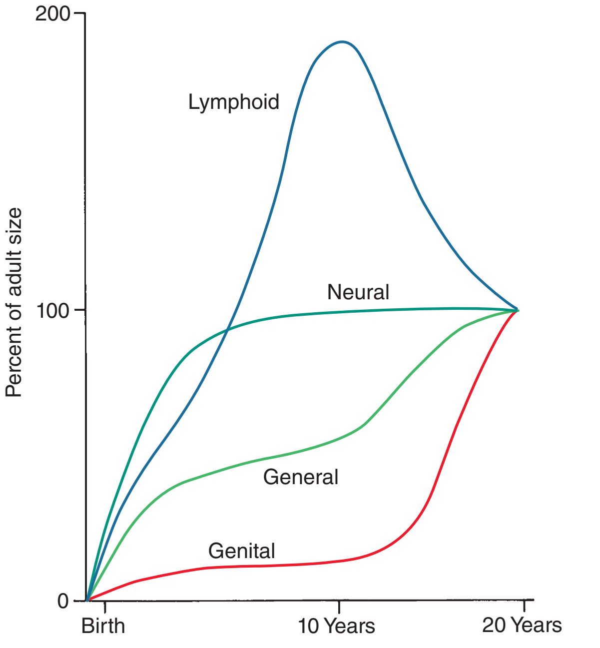


• **Fig. 2.1** Schematic representation of the changes in overall body proportions during normal growth and development. After the third month of fetal life, the proportion of total body size contributed by the head and face steadily declines. (Redrawn from Robbins WJ, et al. *Growth*. Yale University Press; 1928.)

proportionality, usually of a complex set of proportions rather than just a single proportional relationship. Pattern in growth also represents proportionality, but in a still more complex way, because it refers not just to a set of proportional relationships at a point in time but to the change in these proportional relationships over time. In other words, the physical arrangement of the body at any one time is a pattern of spatially proportioned parts. However, there is a higher level pattern, the pattern of growth, which refers to the changes in these spatial proportions over time.

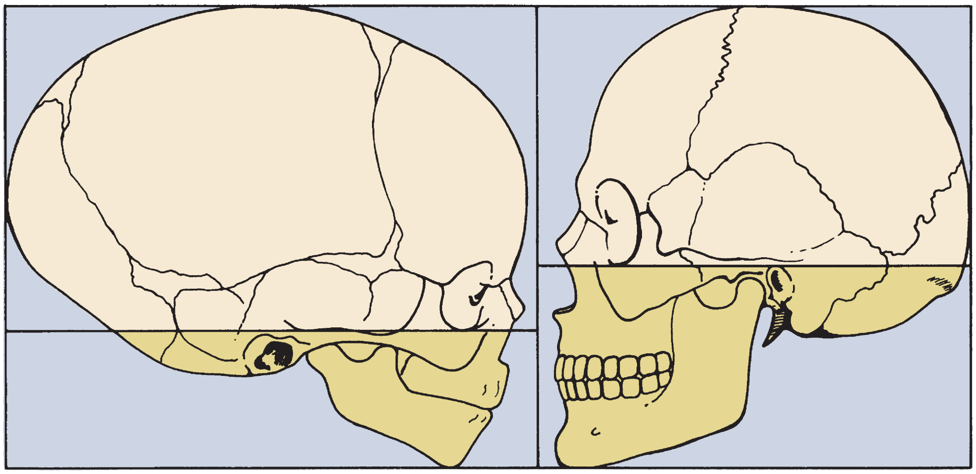
Fig. 2.1 illustrates the change in overall body proportions that occurs during normal growth and development. In fetal life, at about the third month of intrauterine development, the head takes up almost 50% of the total body length. At this stage, the cranium is large relative to the face and represents more than half the total head. In contrast, the limbs are still rudimentary, and the trunk is underdeveloped. By the time of birth, the trunk and limbs have grown faster than the head and face, so that the proportion of the entire body devoted to the head has decreased to about 30%. The overall pattern of growth thereafter follows this course, with a progressive reduction of the relative size of the head to about 12% in the adult. At birth the legs represent about one-third of the total body length, whereas in the adult they represent about half. As Fig. 2.1 illustrates, there is more growth of the lower limbs than the upper limbs during postnatal life. All of these changes, which are a part of the normal growth pattern, reflect the "cephalocaudal gradient of growth." This simply means that there is an axis of increased growth extending from the head toward the feet.

Another aspect of the normal growth pattern is that not all the tissue systems of the body grow at the same rate (Fig. 2.2). Obviously, as the relative decrease in head size after birth shows, the muscular and skeletal elements grow faster than the brain and central nervous system. The overall pattern of growth reflects the growth of the various tissues making up the whole organism. To put it differently, one reason for gradients of growth is that different tissue systems that grow at different rates are concentrated in various parts of the body.



• **Fig. 2.2** Scammon's curves for the growth of the four major tissue systems of the body. As the graph indicates, growth of the neural tissues is nearly complete by 6 or 7 years of age. General body tissues, including muscle, bone, and viscera, show an S-shaped curve, with a definite slowing of the rate of growth during childhood and an acceleration at puberty. Lymphoid tissues proliferate far beyond the adult amount in late childhood and then undergo involution at the same time that the growth of the genital tissues accelerates rapidly. From Scammon RD. The measurement of the body in childhood. In: Harris JA, ed. *The Measurement of Man*. University of Minnesota Press; 1930.

Even within the head and face, the cephalocaudal growth gradient strongly affects proportions and leads to changes in proportion with growth (Fig. 2.3). When the skull of a newborn infant is compared proportionally with that of an adult, it is easy to see that the



• **Fig. 2.3** Changes in proportions of the head and face during growth. At birth, the face and jaws are relatively underdeveloped compared with their extent in the adult. As a result, there is much more growth of facial than cranial structures postnatally. (Redrawn from Lowery GH. *Growth and Development of Children*. 6th ed. Year Book Medical Publishers; 1973.)

infant has a relatively much larger cranium and a much smaller face. This change is an important aspect of the pattern of facial growth. Not only is there a cephalocaudal gradient of growth within the body, but there is also one within the face. From that perspective, it is not surprising that the mandible, being farther away from the brain, tends to grow more and later than the maxilla, which is closer.

An important aspect of a pattern is its predictability. Patterns repeat, whether in the organization of different-colored tiles in the design of a floor or in skeletal proportions changing over time. The proportional relationships within a pattern can be specified mathematically, and the only difference between a growth pattern and a geometric one is the addition of a time dimension. Thinking about patterns in this way allows one to be more precise in defining what constitutes a change in pattern. Change, clearly, would denote an alteration in the predictable pattern of mathematical relationships. A change in growth pattern would indicate some alteration in the expected changes in body proportions.

A second important concept in the study of growth and development is variability. Obviously, all people are not alike in the way that they grow, as in everything else. It can be difficult but clinically very important to decide whether an individual is merely at the extreme of the normal variation or falls outside the normal range.

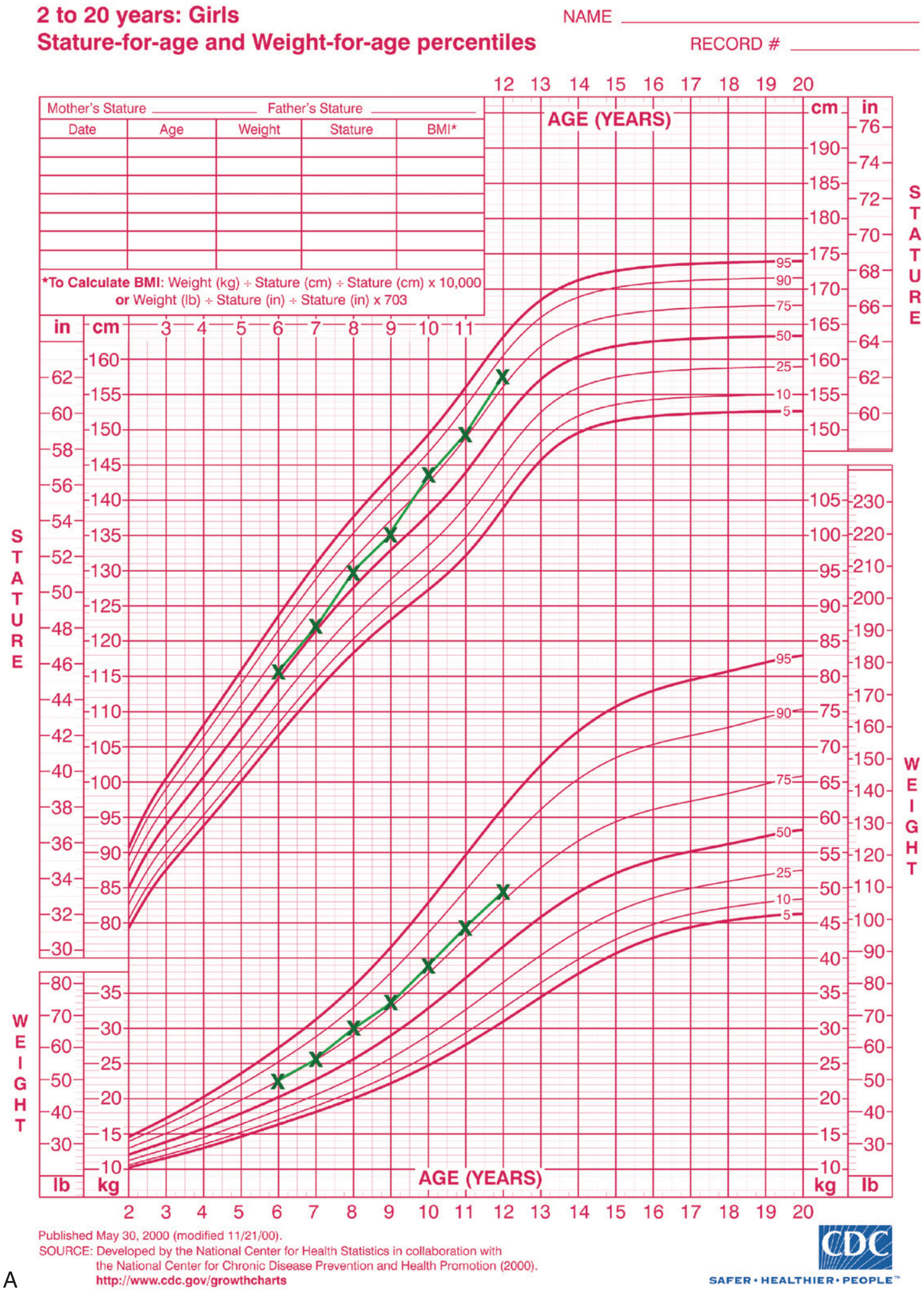
Rather than categorizing growth as normal or abnormal, it is more useful to think in terms of deviations from the usual pattern and to express variability quantitatively. One way to do this is to evaluate a given child relative to peers on a standard growth chart (Fig. 2.4). Although charts of this type are commonly used for height and weight, the growth of any part of the body can be plotted in this way. The "normal variability," as derived from large-scale studies of groups of children, is shown by the solid lines on the graphs. An individual who stood exactly at the midpoint of the normal distribution would fall along the 50% line of the graph. One who was larger than 90% of the population would plot above the 90% line; one who was smaller than 90% of the population would plot below the 10% line.

These charts can be used in two ways to determine whether the growth is normal or abnormal. First, the location of an individual relative to the group can be established. A general guideline is that a child who falls outside the range of 97% of the population should receive special study before being accepted as just an extreme of the normal population. Second and perhaps more important, growth charts can be used to follow a child over time to evaluate whether there is an unexpected change in the growth pattern. Pattern implies predictability. For the growth charts, this means that a child's growth should be generally plotted along the same percentile line at all ages. If the percentile position of an individual relative to their peer group changes, especially if there is a marked change (see Fig. 2.4B), the clinician should suspect some growth abnormality and should investigate further. Inevitably, there is a gray area at the extremes of normal variations, at which it is difficult to determine if the growth is normal.

A final major concept in physical growth and development is timing. Variability in growth arises in several ways: from normal variation, from influences outside the normal experience (e.g., serious illness), and from timing effects. Variation in timing arises because the same event happens for different individuals at different times—or, viewed differently, the biological clocks of different individuals are set differently.

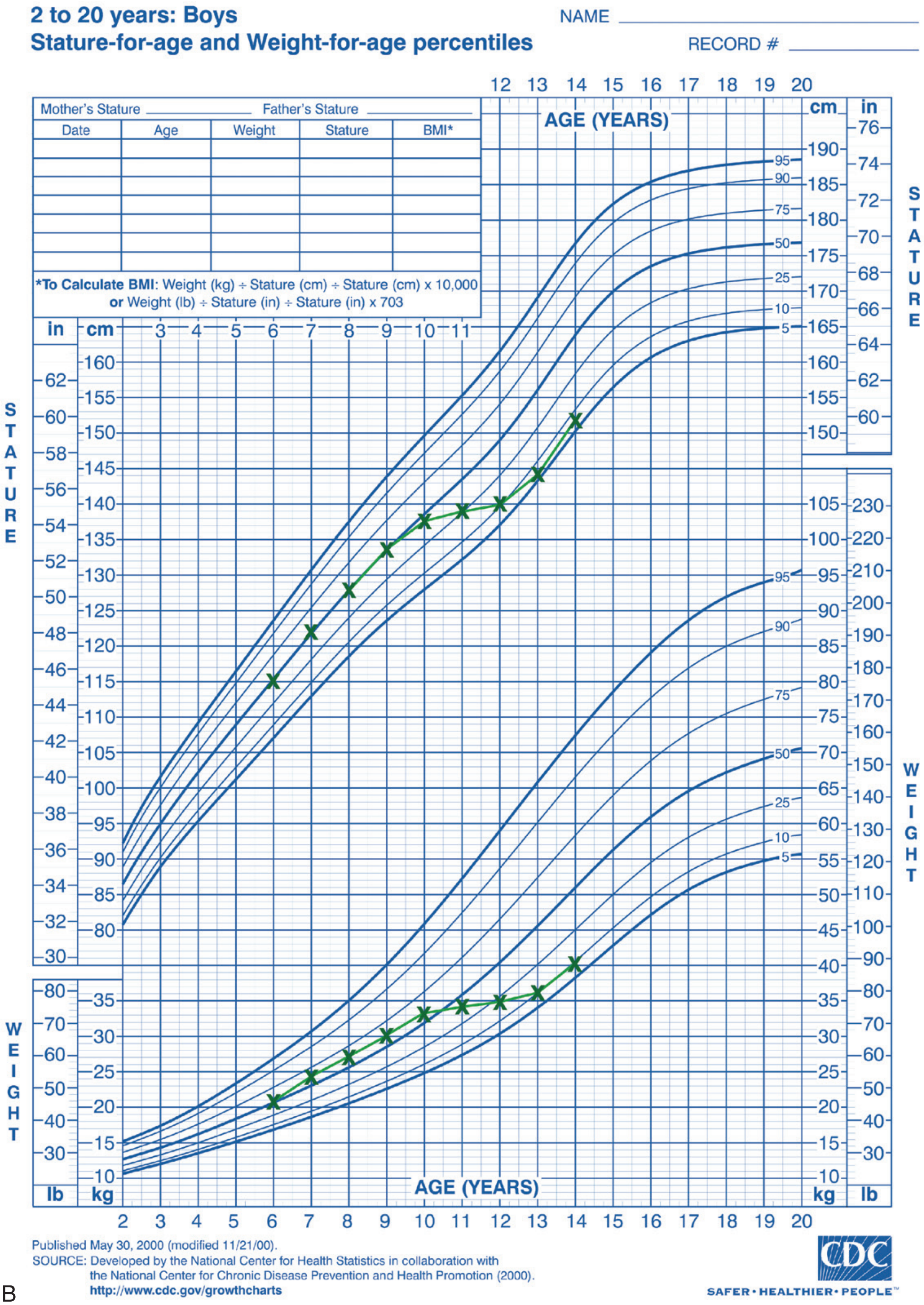
Variations in growth and development because of timing are particularly evident in human adolescence. Some children grow rapidly and mature early, completing their growth quickly and thereby appearing on the high side of developmental charts until their growth ceases and their contemporaries begin to catch up. Others grow and develop slowly and so appear to be behind, even though, given time, they will catch up with and even surpass children who once were larger. All children undergo a spurt of growth at adolescence, which can be seen more clearly by plotting changes in height or weight (Fig. 2.5), but the growth spurt occurs at different times in different individuals.

Growth effects because of timing variation can be seen particularly clearly in girls, in whom the onset of menstruation (menarche) gives an excellent indicator of the arrival of sexual maturity. The process of sexual maturation is accompanied by a spurt in growth. When the growth velocity curves for early-, average-, and late-maturing girls are compared in Fig. 2.6, the marked differences in size among these girls during growth are apparent. At age 11, the early-maturing girl is already past the peak of the adolescent growth spurt, whereas the late-maturing girl has not even begun to grow rapidly. This sort of timing variation occurs in many aspects of both growth and development and can be an important contributor to variability.



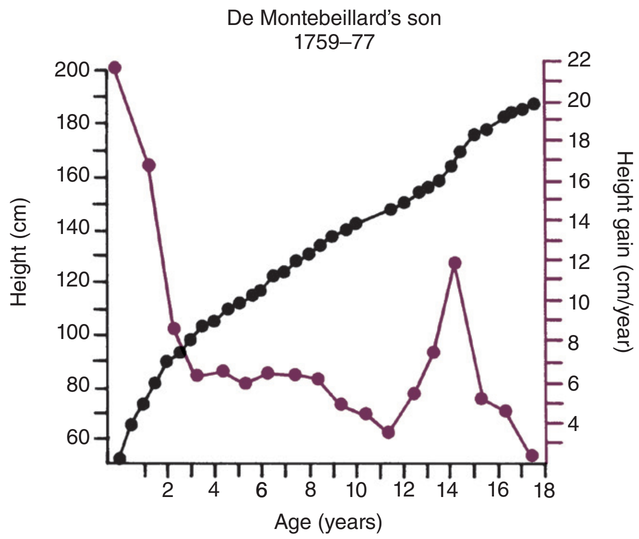
A

• Fig. 2.4 (A) Growth of a normal girl plotted on the chart for females. Note that this girl remained at about the 75th percentile for height and weight over this entire period of observation. *Continued*

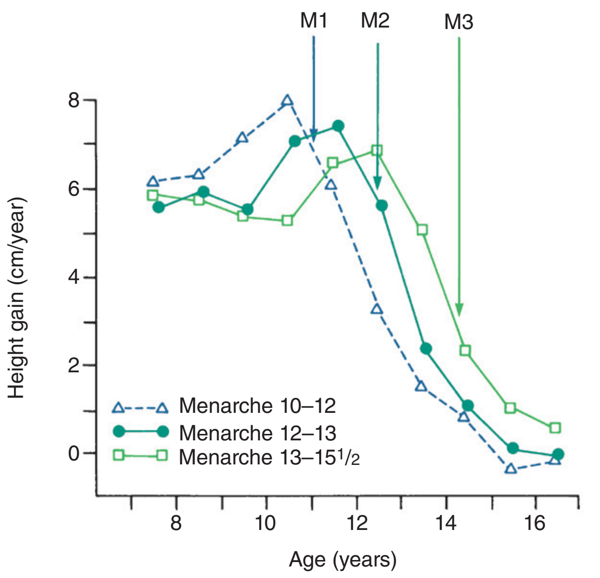


B

• Fig. 2.4, cont'd (B) Growth of a boy who developed a medical problem that affected growth, plotted on the male chart. Note the change in pattern (crossover of lines on the chart) between ages 10 and 11. This reflects the impact of serious illness beginning at that time, with partial recovery after age 13 but a continuing effect on growth. (Data from Hamill PV, et al. National Center for Health Statistics, 1979; charts developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion, published May 30, 2000, revised November 21, 2000—the latest available) (Charts available from http://www.cdc.gov/growthcharts/.)

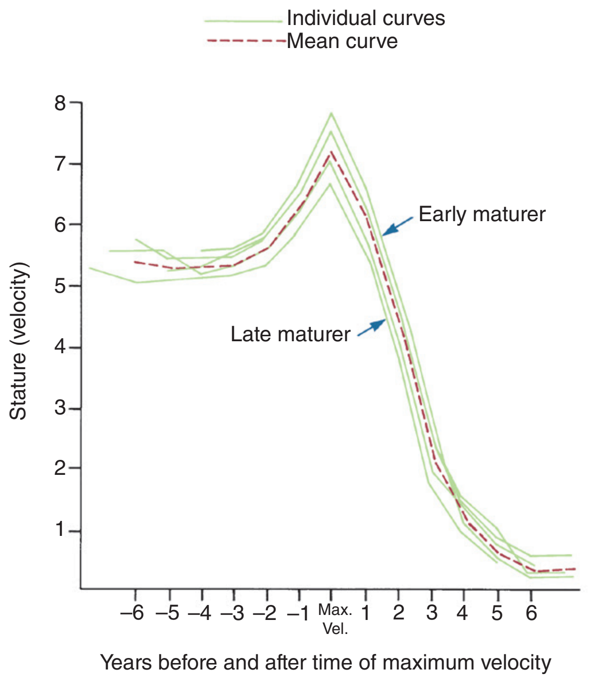


• **Fig. 2.5** Growth can be plotted in either height or weight at any age *(black line)* or the amount of change in any given interval (*maroon line*, showing the same data as the *black line*). A curve like the black line is called a *distance curve*, whereas the maroon line is a *velocity curve*. Plotting velocity rather than distance makes it easier to see when accelerations and decelerations in the rate of growth occurred. These data are for the growth of one individual, the son of a French aristocrat in the late 18th century, whose growth followed the typical pattern. Note the acceleration of growth at adolescence, which occurred for this individual at about age 14. (Data from Scammon RE. The first seriatim study of human growth. *Am J Phys Anthropol*. 1927;10(3):329-336.)



• **Fig. 2.6** Growth velocity curves for early-, average-, and late-maturing girls. It is interesting to note that the earlier the adolescent growth spurt occurs, the more intense it appears to be. Obviously, at age 11 or 12, an early-maturing girl would be considerably larger than one who matured late. In each case, the onset of menstruation (menarche) (*M1, M2*, and *M3*) came after the peak of growth velocity.

Although age is usually measured chronologically as the amount of time since birth or conception, it is also possible to measure age biologically, in terms of progress toward various developmental markers or stages. Timing variability can be reduced by using developmental age rather than chronologic age as an expression of an individual's growth status. For instance, if data for gain in height for girls are replotted, using maximum height velocity as a reference time point (Fig. 2.7), it is apparent that girls who mature early, at an average time, or late really follow a very similar growth pattern. This graph substitutes the stage of growth for chronologic age to produce a biological time scale and shows that the pattern is expressed at different times chronologically but not physiologically. The effectiveness of biological or developmental age in reducing timing variability makes this approach useful in evaluating a child's growth status.



• **Fig. 2.7** Velocity curves for four girls with quite different times of menarche, replotted using menarche as a zero time point. It is apparent that the growth pattern in each case is quite similar, with almost all of the variations resulting from timing.

Methods for Studying Physical Growth

Before beginning the examination of growth data, it is important to have a reasonable idea of how the data were obtained. There are two basic approaches to studying physical growth. The first is based on techniques for measuring living animals (including humans), with the implication that the measurement itself does no harm and that the animal will be available for additional measurements at another time. The second approach uses experiments in which growth is manipulated in some way. This implies that the subject of the experiment will be available for study in some detail, and the detailed study may be destructive. For this reason, such experimental studies are largely restricted to nonhuman species.

Measurement Approaches

Acquiring Measurement Data

Craniometry

The first of the measurement approaches for studying growth, with which the science of physical anthropology began, is craniometry, based on measurements of skulls found among human skeletal remains. Craniometry was originally used to study the Neanderthal and Cro-Magnon peoples whose skulls were found in European caves in the 18th and 19th centuries. From such skeletal material, it has been possible to piece together a great deal of knowledge about extinct populations and to get some idea of their pattern of growth by comparing one skull with another. Craniometry has the advantage that rather precise measurements can be made on dry skulls; it has the important disadvantage for growth studies that, by necessity, all these growth data must be cross-sectional. *Cross-sectional* means that although different ages are represented in the population, the same individual can be measured at only one point in time.

Anthropometry

It is also possible to measure skeletal dimensions in living individuals. In this technique, called *anthropometry*, various landmarks established in studies of dry skulls are measured in living individuals simply by using soft tissue points overlying these bony landmarks. For example, it is possible to measure the length of the cranium from a point at the bridge of the nose to a point at the greatest convexity of the rear of the skull. This measurement can be made on either a dried skull or a living individual, but results would be different because of the soft tissue thickness overlying both landmarks. Although the soft tissue introduces variation, anthropometry does make it possible to follow the growth of an individual directly, making the same measurements repeatedly at different times. This produces longitudinal data: repeated measures of the same individual. The best data of that type come from Farkas's anthropometric studies in the late 20th century, which provided valuable new data for human facial proportions and their changes from childhood to adolescence and adult life.[1]

Cephalometric Radiology

The third measurement technique, cephalometric radiology, is of considerable importance not only in the study of growth but also in the clinical evaluation of orthodontic patients. The technique depends on precisely orienting the head before making a radiograph, with equally precise control of magnification. This approach can combine the advantages of craniometry and anthropometry. It allows a direct measurement of bony skeletal dimensions because the bone can be seen through the soft tissue covering in a radiograph, but it also allows the same individual to be followed over time. Growth studies are done by superimposing a tracing or digital model of a later cephalogram on an earlier one, so that the changes can be measured. Both the locations and amounts of growth can be observed in this way (Fig. 2.8). Cephalometric superimposition techniques are described in detail in Chapter 6.

The disadvantage of a standard cephalometric radiograph is that it produces a two-dimensional (2D) representation of a three-dimensional (3D) structure, and so, even with precise head positioning, not all measurements are possible. To some extent, this can be overcome by making more than one radiograph at different orientations and using triangulation to calculate oblique distances. The general pattern of craniofacial growth was known from craniometric and anthropometric studies before cephalometric radiography was invented, but much of the current picture of craniofacial growth is based on cephalometric studies.

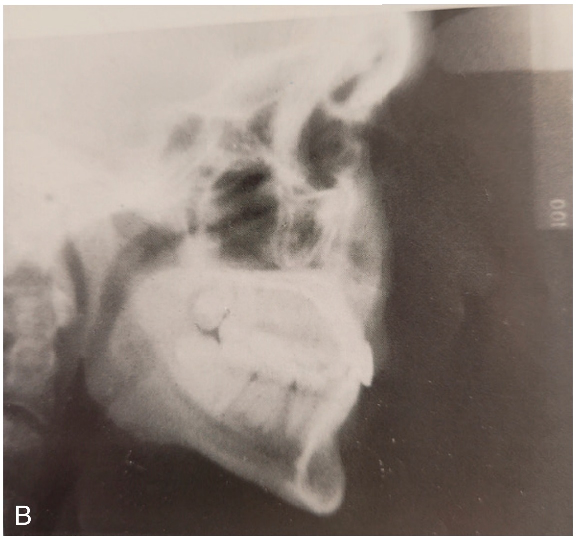
Three-Dimensional Imaging

New information is now being obtained with the application of 3D imaging techniques. Computed axial tomography (CAT or, more commonly, computed tomography [CT]) allows 3D reconstructions of the cranium and face, and this method has been applied in the last 35 years to plan surgical treatment for patients with facial deformities (Fig. 2.9). Recently, cone beam computed tomography (CBCT) rather than axial CT has been applied to scans of the head and face. This significantly reduces both the radiation dose and the cost. CBCT allows scans of patients with radiation exposure that are much closer to the dose from cephalograms. CBCT also reduces flaws within 2D imaging such as head position, superimposition of anatomical structures, and magnification errors. The superimposition of 3D images is much more difficult than the superimpositions used with 2D cephalometric radiographs, but methods have been developed to address the time and difficulty involved (Fig. 2.10).[2]

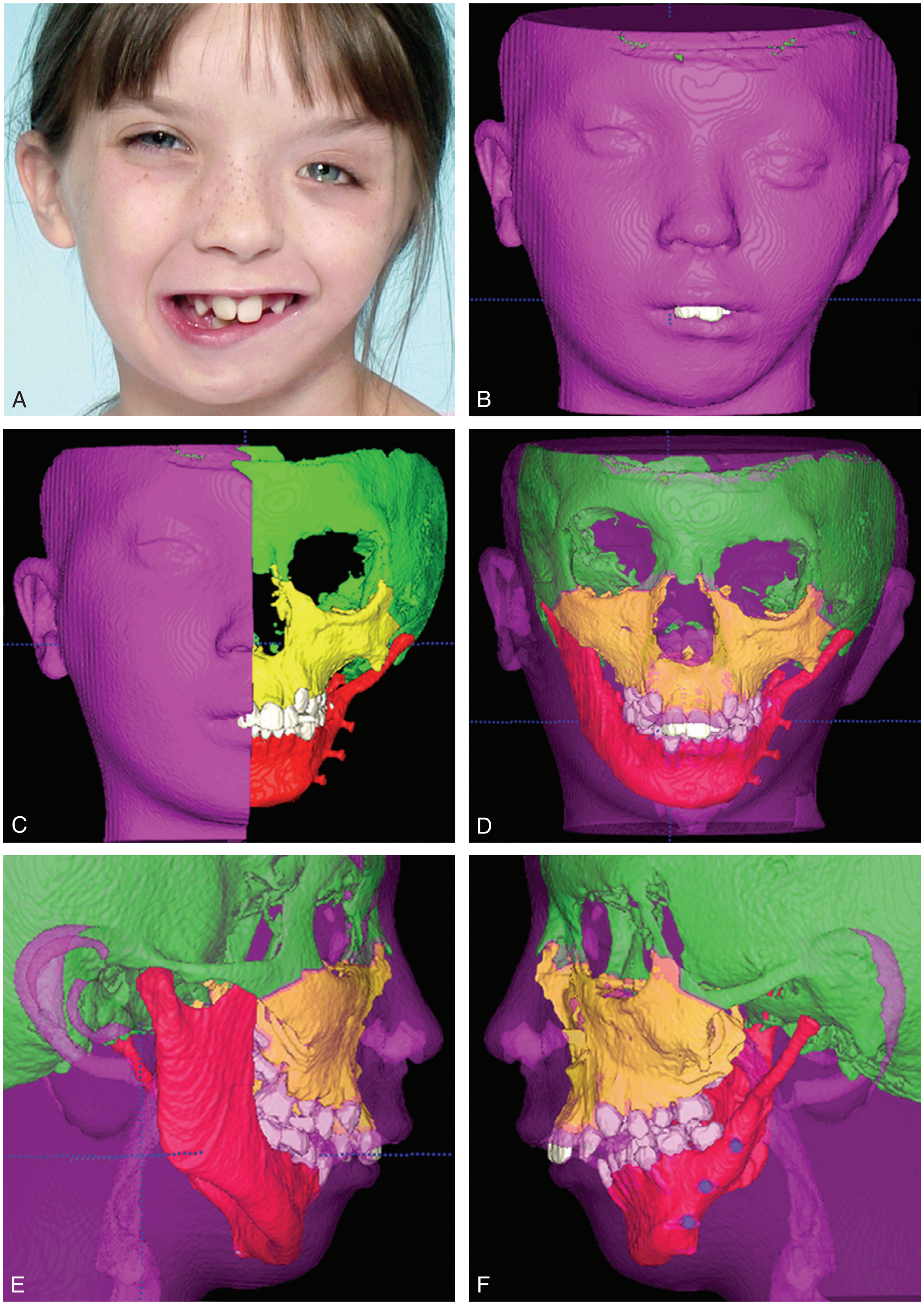
Magnetic resonance imaging (MRI) also provides 3D images that can be useful in studies of growth, with the advantage that there is no radiation exposure with this technique. This method has already been applied to the analysis of the growth changes produced by functional appliances,[3] but it shows soft tissues more clearly than hard tissues, just the reverse of radiographic images.

Three-dimensional photography now makes possible much more accurate measurements of facial soft tissue dimensions and changes (Fig. 2.11). A more detailed examination of 3D changes

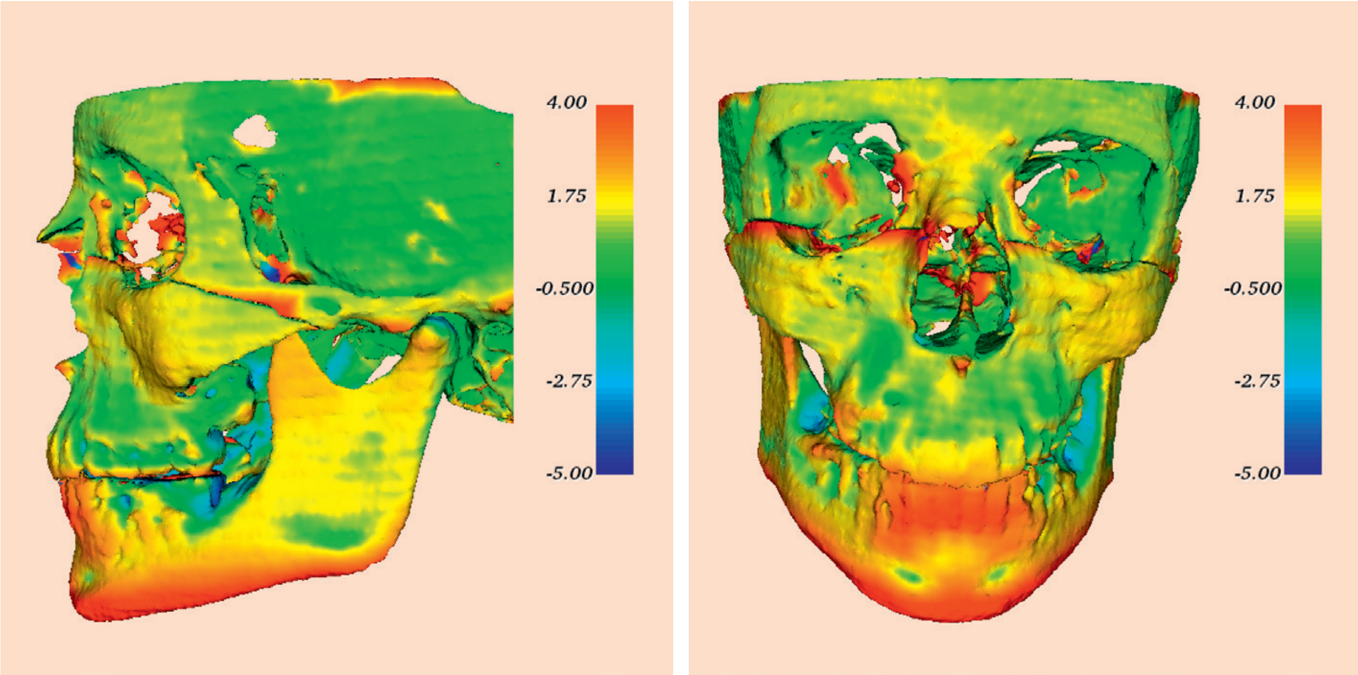
A


B


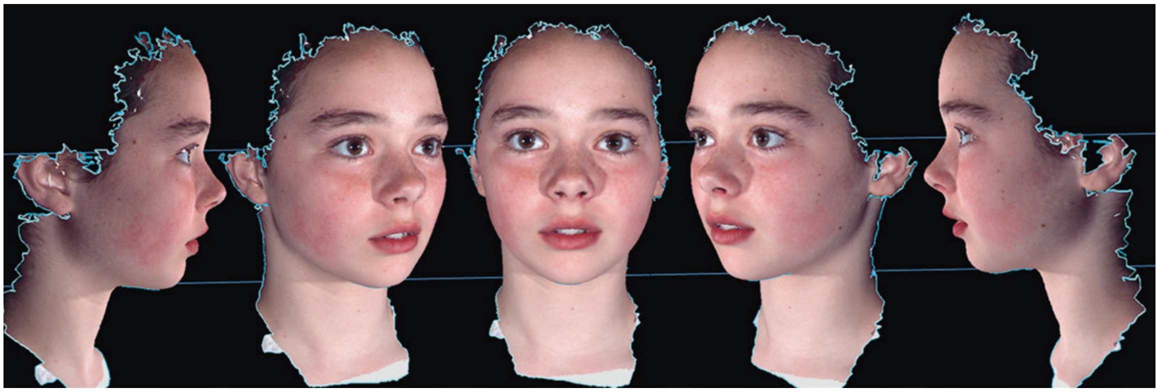
• **Fig. 2.8** (A) A cephalometric radiograph merits this name because of the use of a head-positioning device to provide precise orientation of the head. This means that valid comparisons can be made between external and internal dimensions in members of the same population group or that the same individual can be measured at two points in time because the head orientation is reproducible. (B) This radiograph (a cephalogram) was taken in natural head position (NHP) (see Chapter 6 for a description of this head-positioning technique).



• **Fig. 2.9** Computed tomography (CT) scans are the best way to determine the details of skeletal deformities. These views of a 9-year-old girl (A) with severe hemifacial microsomia (and previous surgical treatment to build up the affected side of the mandible) illustrate that CT scans can show both skin contours and bony relationships from any aspect. Color can be added to different structures to make it easier to visualize them (B), and surface layers can be made transparent (as in C–F) to reveal the skeletal structures beneath. Views of this type greatly facilitate surgical treatment planning. (Courtesy Dr. L. Cevidanes.)



• **Fig. 2.10** Superimposition of computed tomography (CT) images is much more difficult than superimposition of cephalometric tracings but more accurately detects the amount of change and can be used to see changes in exquisite detail. These images are superimposed on the cranial base; a color map is used to show the change from the initial image of an adolescent with a normal growth pattern at age 12 to the same individual at age 14. In the color map, green shows areas of little or no change; deepening shades of red indicate 3–4 mm of movement away from the cranial base; and shades of blue (seen here only on the front of the mandibular ramus) indicate areas that moved closer to the cranial base. The map shows the downward and forward movement of the mandible at a time, as you would expect, of more mandibular than maxillary growth and resorption of the front of the ramus that lengthens the body of the mandible to provide space for eruption of the second molars. Note that the zygomatic arches and the maxillary teeth moved forward a small amount while the bony area just above the maxilla stayed largely in the same place. Understanding growth changes from carefully examining superimposition images such as these is much easier than deciphering the meaning of a series of measurements and gives a broader view of the skeletal changes due to growth.

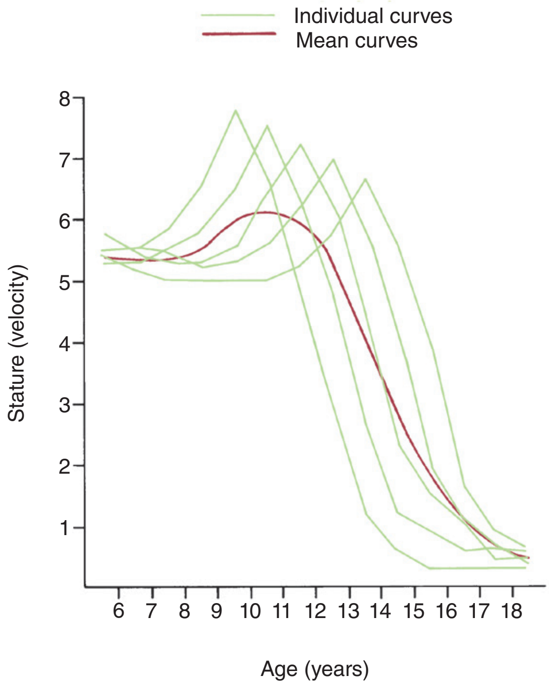


• **Fig. 2.11** Images from a single photograph with a 3dMD camera. Both profile and oblique and frontal views can be captured at the same head position, and measurements of soft tissue dimensions and proportions can be made with great accuracy at any orientation of the face, which makes a camera that provides such three-dimensional views a valuable research tool.

in growing patients almost surely will add to current knowledge of growth patterns in the near future.

Analysis of Measurement Data

Both anthropometric and cephalometric data can be expressed cross-sectionally rather than longitudinally. Obviously, it would be much easier and quicker to do a cross-sectional study, gathering data once for any individual and including subjects of different ages, rather than spending many years on a study in which the same individuals are measured repeatedly. For this reason, most studies are cross-sectional. When this approach is used, however, variability within the sample can conceal details of the growth



• **Fig. 2.12** If growth velocity data for a group of individuals with different timings for their adolescent growth spurt are plotted on a chronologic scale, it is apparent that the average curve is not an accurate representation of the pattern of growth for many individuals. This smoothing of individual variation is a characteristic of cross-sectional data and a major limitation in the use of the cross-sectional method for studies of growth. Only by following individuals through time in a longitudinal study is it possible to see the details of growth patterns.

pattern, particularly when there is no correction for timing variation (Fig. 2.12). Fluctuations in the growth curve that may occur for nearly every individual would be seen in a cross-sectional study only if they occurred at the same time for each person, which is unlikely. Longitudinal studies are efficient in the sense that a great deal of information can be gained from a relatively small number of subjects, fewer than would be needed in a cross-sectional study. In addition, the longitudinal data highlight individual variations, particularly variations caused by timing effects.

Measurement data can be presented graphically in a number of different ways, and frequently it is possible to clarify growth changes by varying the method of display. For example, we have already seen that growth data can be shown either by plotting the size attained as a function of age, which is called a "distance" curve, or as a "velocity" curve, showing not the total length but the increment added each year (see Fig. 2.5). Changes in the rate of growth are much more easily seen in a velocity curve.

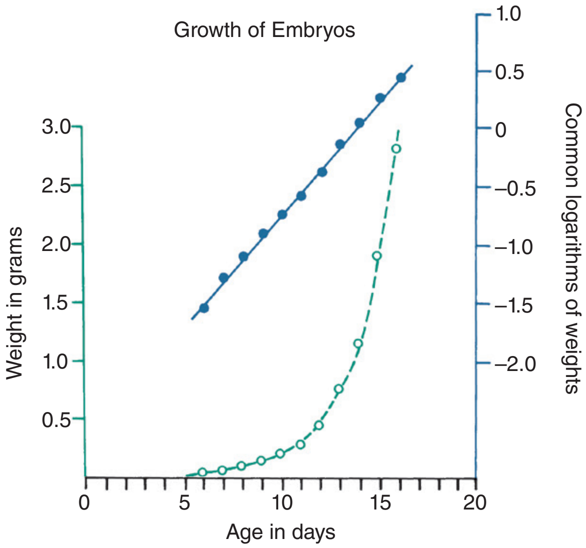
Various other mathematical transformations can be used with growth data to make them easier to understand. For instance, the growth in weight of any embryo at an early stage follows a logarithmic or exponential curve because the growth is based on the division of cells; the more cells there are, the more cell divisions can occur. If the same data are plotted using the logarithm of the weight, a straight-line plot is attained (Fig. 2.13). This demonstrates that the rate of multiplication for cells in the embryo remains more or less constant.

More complex mathematical transformations were used many years ago by D'Arcy Thompson[4] to reveal similarities in proportions and growth changes that had not previously been suspected (Fig. 2.14). To correctly interpret data after mathematical transformation, it is important to understand how the data were transformed, but the approach is a powerful one in clarifying growth concepts, and Thompson's classic presentation remains stimulating reading.

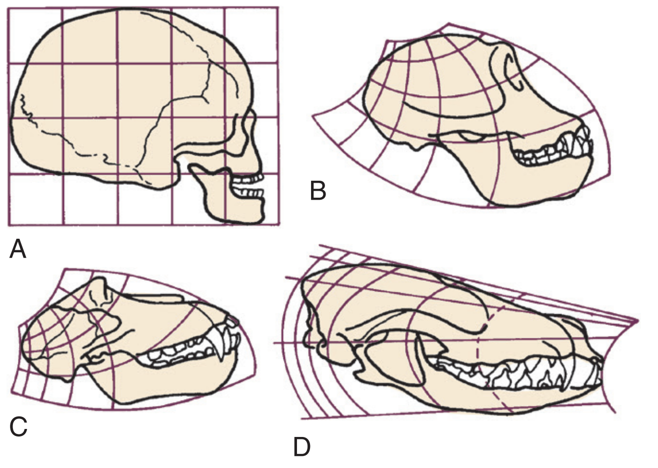
Experimental Approaches

Vital Staining

Much has been learned about skeletal growth using the technique called *vital staining*, in which dyes that stain mineralizing tissues (or occasionally, soft tissues) are injected into an animal. These dyes remain in the bones and teeth and can be detected later after



• **Fig. 2.13** Data for the increase in weight of early embryos, with the raw data plotted in green and the same data plotted after logarithmic transformation in blue. At this stage the weight of the embryo increases dramatically, but, as shown by the straight line after transformation, the rate of multiplication of individual cells remains fairly constant. When more cells are present, more divisions can occur, and the weight increases faster. (From Lowery GH. *Growth and Development of Children*. 8th ed. Year Book Medical Publishers; 1986.)

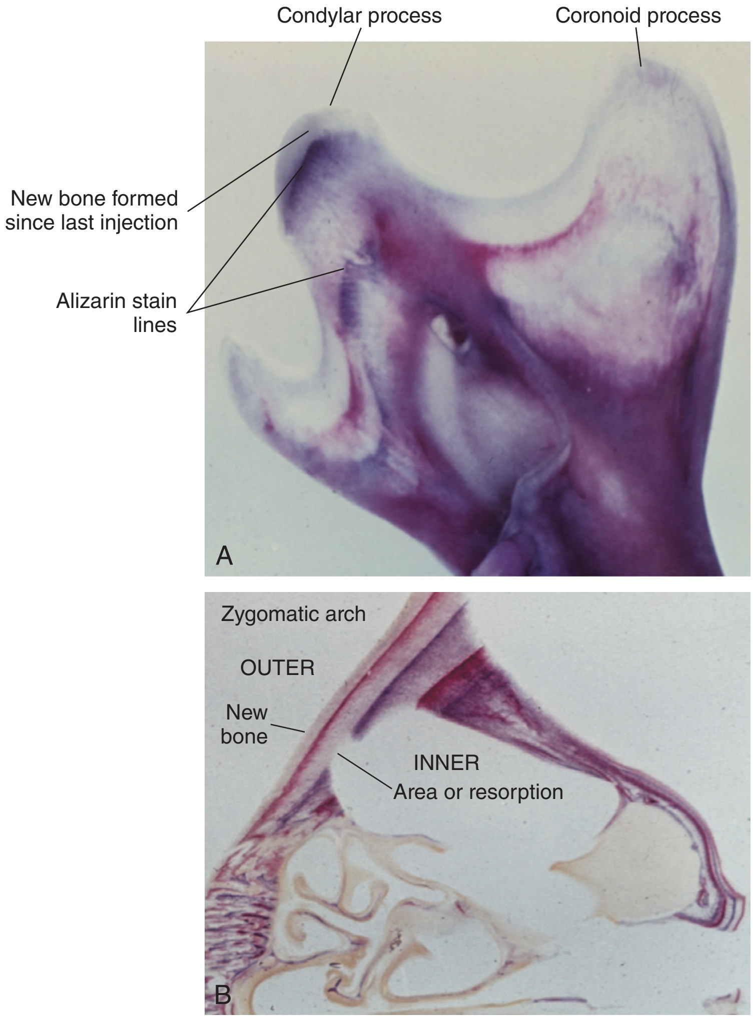


• **Fig. 2.14** In the early 1900s D'Arcy Thompson showed that mathematical transformation of a grid could account for the changes in the shape of the face from man (A) to chimpanzee (B), monkey (C), dog (D), or other animals. Application of this method revealed previously unsuspected similarities among various species. (Redrawn from Thompson DW. *On Growth and Form*. Cambridge University Press; 1961.)

death. This method was originated by the great English anatomist John Hunter in the 18th century. Hunter observed that the bones of pigs that occasionally were fed textile waste were often stained in an interesting way. He discovered that the active agent was a dye called *alizarin*, which is still used for vital staining studies. Alizarin reacts strongly with calcium at sites where bone calcification occurs. Because these are the sites of active skeletal growth, the dye marks the locations at which active growth was occurring when it was injected. Bone models rapidly, and areas from which bone is being removed can also be identified by the fact that vital stained material has been removed from these locations (Fig. 2.15). Highly detailed vital staining studies of bony changes in craniofacial development in experimental animals, from work done at the US National Institute of Dental and Craniofacial Research, are available.[5]

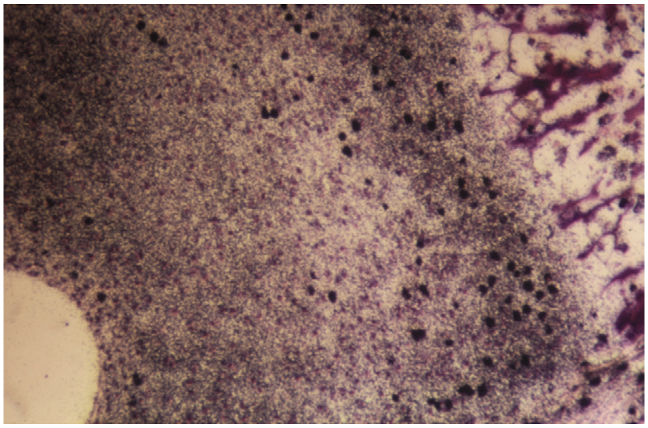
Although studies using vital stains are not possible in humans, vital staining can occur inadvertently. Many children born in the late 1950s and early 1960s were treated for recurrent infections with the antibiotic tetracycline. It was discovered too late that tetracycline is an excellent vital stain that binds to calcium at growth sites in the same way as alizarin. The discoloration of incisor teeth that results from tetracycline given when the teeth are mineralizing has been a serious esthetic problem for some individuals (Fig. 2.16). Although this does not commonly occur now, it is still seen occasionally.

With the development of radioactive tracers, it has become possible to use almost any radioactively labeled metabolite that becomes incorporated into the tissues as a sort of vital stain. The location is detected by the weak radioactivity given off at the site where the material was incorporated. The gamma-emitting isotope technetium 99 m (^{99m}Tc) can be used to detect areas of rapid bone growth in humans, but these images are more useful in the diagnosis of localized growth problems such as condylar hyperplasia (see Chapter 6) than for studies of growth patterns. For most studies of growth, radioactively labeled materials in the tissues of experimental animals are detected by the technique of



• **Fig. 2.15** (A) The mandible of a growing rat that received four injections of alizarin (red-blue-red-blue) at 2-week intervals and was killed 2 weeks after the last injection (so that the bone formed since then is white). Modeling of the bone as it grows blurs some of the lines of intensely colored bone created by each injection, but the red-blue sequential lines in the condylar process can be seen clearly. (B) Section through the zygomatic arch from the same animal. The zygomatic arch grows outward by apposition of bone on the outer surface and removal from the inner surface. The interruptions in the staining lines on the inner surface clearly show the areas where the bone is being removed. What was once the outer surface of the zygomatic arch becomes the inner surface a relatively short time later and then is removed.

• **Fig. 2.16** Tetracycline staining in the teeth of a boy who received large doses of tetracycline because of repeated upper respiratory infections in early childhood. From the location of the staining, it is apparent that tetracycline was not administered in infancy but was given in large doses beginning when the crowns of the central incisors were about half formed or at approximately 30 months.

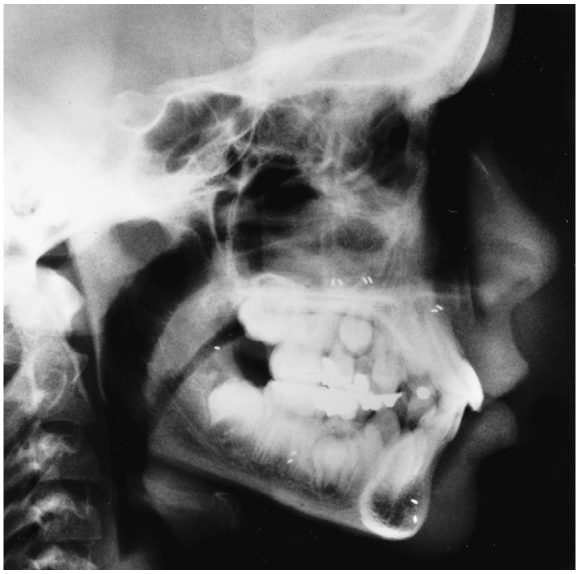


• **Fig. 2.17** An autoradiograph of fetal rat bones growing in organ culture, with carbon 14 (^{14}C)–proline and tritium (^{3}H)–thymidine incorporated in the culture medium. Thymidine is incorporated into DNA, which is replicated when a cell divides, so labeled nuclei are those of cells that underwent mitosis in culture. Because proline is a major constituent of collagen, cytoplasmic labeling indicates areas where proline was incorporated, primarily into extracellularly secreted collagen.

autoradiography, in which a film emulsion is placed over a thin section of tissue containing the isotope and is then exposed in the dark by the radiation. After the film has been developed, the location of the radiation that indicates where growth is occurring can be observed by looking at the tissue section through the film (Fig. 2.17).

Implant Radiography

Another experimental method applicable to studies of humans is implant radiography. In this technique, inert metal pins are placed in bones anywhere in the skeleton, including the face and jaws. These metal pins are well tolerated by the skeleton, become permanently incorporated into the bone without causing any problems, and are easily visualized on a cephalogram (Fig. 2.18). If they are placed in the jaws, a considerable increase in the accuracy of a longitudinal cephalometric analysis of growth patterns can be achieved. This method of study was developed by



• **Fig. 2.18** Lateral cephalometric radiograph from the archives of Björk's implant studies showing a subject with six maxillary and five mandibular tantalum implants. (Courtesy Department of Orthodontics, University of Copenhagen, Denmark.)

Professor Arne Björk and coworkers at the Royal Dental College in Copenhagen, Denmark, and was used extensively by workers there (see Chapter 4). It provided important new information about the growth pattern of the jaws.

With 21st-century technology, precise evaluation of dentofacial growth in humans using implant cephalograms has largely been superseded by 3D imaging via CBCT or MRI, but it still can be helpful to use implants to provide landmarks for superimposition.

Genetic Influences on Growth

Rapid advances in molecular genetics are providing new information about growth, its control, and links to the development of orthodontic problems. It has now been shown that homeobox *Msx* and *Dlx* genes, which are known to be critically important in the establishment of the body plan, pattern formation, and morphogenesis, are expressed differentially not only in the development of the teeth but also in the growth of the mandible. Msx-1 predominates in tooth formation and is expressed in mesenchyme during tooth development, whereas Msx-2 is expressed in both epithelial and mesenchymal components of the developing tooth germ.[6–8] Dlx-1 and Dlx-2 are expressed in the dental mesenchyme and in the epithelium of the maxillary and mandibular arch mesenchyme, and other homeobox gene groups have been shown to play a role in dental and facial development.[9] An association between a specific genotype for muscle myofibril anchor proteins and class II and deep bite malocclusions has been demonstrated.[10]

The proper function of families of growth factors and their cognate receptors is essential in regulating embryonic processes of cell growth and organ development, as well as a myriad postnatal processes that include growth, wound healing, bone remodeling, and homeostasis. The US National Institute of Dental and Craniofacial Research has recently announced the establishment of a consortium to accelerate the understanding of craniofacial developmental biology through interactive projects including global and specific gene expression patterns, genome-wide association patterns, and transcriptional profiling over the course of embryonic and postnatal development in animal models and humans.[11] Essentially, this is a project toward the use of "big data" to better understand complex genetic interactions. It seems likely that interactive research of this type by groups of investigators will lead to the development of gene therapy for developmental problems.

Interaction between different tissues within the craniofacial complex creates yet another level of regulation of growth and development. One example of this is the convergence of the development of the muscles that attach to the mandible and the bony areas to which they attach. Although many genes are involved in determining mandibular size, genetic alterations in muscle development and function translate into changes in the forces on areas of bone where muscles attach, and this leads to the modification of skeletal areas such as the coronoid process and gonial angle area of the mandible. Genetic alterations that affect muscle also would affect these skeletal areas. To understand this, it is necessary to identify the specific genes involved and to deduce how their activity is modified, but it is already apparent that gene expression can be upregulated or downregulated by mechanical stresses.

Another good example of complex, tightly regulated tissue interactions is found in the development of a tooth and the beginning of eruption. Tooth formation begins with the differentiation of ameloblasts (which form the outer enamel layer of the crown of a tooth) and odontoblasts (which form the dentin layer) within the rapidly calcifying alveolar bone, while multipotential cells continue to be present within the forming crown. As crown formation continues, the dental follicle, a fibrous sac containing the developing tooth, signals the differentiation of osteoblasts from precursors and the differentiation of odontoclasts (resorb dentin) and osteoclasts (resorb bone) from monocytes. This process is mediated by a number of signaling factors including CSF-1, MCSP-1, and OPG/RANK/RANKL. Mature osteoclasts resorb bone over the crown allowing the eruptive movement initiated by the dental follicle. The expression of these factors is time sensitive and correlates with the formation of the tooth root, which initiates the occlusal movement characteristic of eruption into the oral cavity.[12]

There has been significant progress in understanding the genetics of human eruption problems, and the identification of a genetic mutation leading to primary failure of eruption (PFE) in 2009 made it possible for the first time to diagnose an orthodontic problem from examination of DNA from a sample of blood or saliva.[13] PFE is discussed further in Chapters 3 and 12.

An exciting prospect for the future is a better understanding of how patients with orthodontic problems that are known to have a genetic component (class III malocclusion being the best example) will respond to treatment. Chromosomal loci associated with class III malocclusion have been identified. There are multiple subtypes of class III, and a necessary first step is better characterization of these phenotypes. Establishing phenotypic markers (distinct clinical characteristics) makes it possible to establish definitive correlations with modes of inheritance and is necessary for linkage studies that will clarify the genetic basis for the problem.

It is unlikely that genetic analysis will ever be applicable to planning treatment for the majority of orthodontic problems, but it could yield valuable information about the best approach to some of the most difficult skeletal malocclusions, predicting how a patient will respond to treatment and perhaps the application of gene therapy to growth problems.

The Nature of Skeletal Growth

At the cellular level, there are only three possibilities for growth. The first is an increase in the size of individual cells, which is referred to as *hypertrophy*. The second possibility is an increase in the number of cells, which is called *hyperplasia*. The third is the *secretion of extracellular material*, thus contributing to an increase in size independent of the number or size of the cells themselves.

In fact, all three of these processes occur in skeletal growth. Hyperplasia is a prominent feature of all forms of growth. Hypertrophy occurs in a few special circumstances but is a less important mechanism than hyperplasia in most instances. Although tissues throughout the body secrete extracellular material, this phenomenon is particularly important in the growth of the skeletal system, where extracellular material later mineralizes.

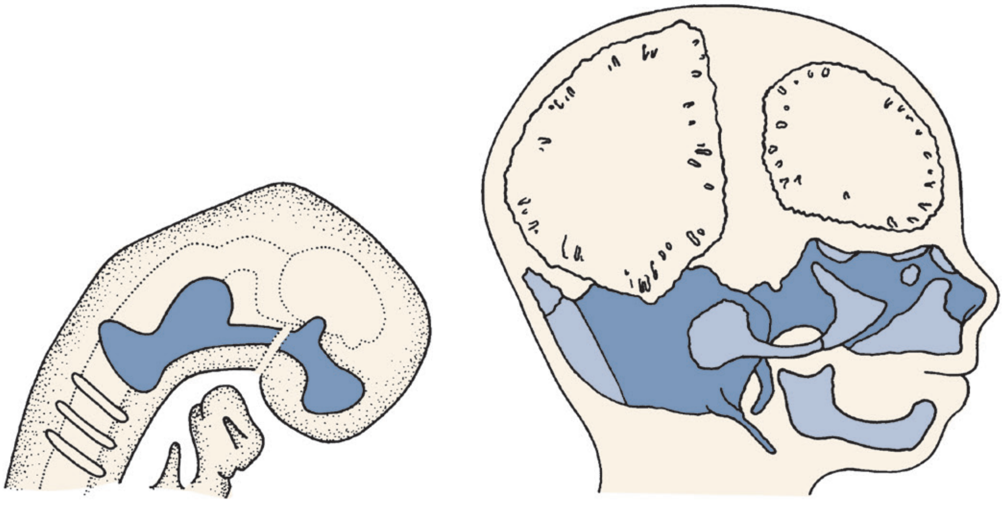
The fact that the extracellular material of the skeleton becomes mineralized leads to an important distinction between the growth of the soft or nonmineralized tissues of the body and the hard or calcified tissues. Hard tissues are bones, teeth, and sometimes cartilages. Soft tissues are everything else. In most instances, cartilage, particularly the cartilage significantly involved in growth, behaves like soft tissue and should be thought of in that group rather than as hard tissue.

Growth of soft tissues occurs by a combination of hyperplasia and hypertrophy. These processes go on everywhere within the tissues, and the result is what is called *interstitial growth*, which simply means that it occurs at all points within the tissue. Although the secretion of extracellular material can also accompany interstitial growth, hyperplasia primarily and hypertrophy secondarily are its characteristics. Interstitial growth is characteristic of nearly all soft tissues and of uncalcified cartilage within the skeletal system.

In contrast, when mineralization takes place so that hard tissue is formed, interstitial growth becomes impossible. Hyperplasia, hypertrophy, and secretion of extracellular material are all still possible, but in mineralized tissues, these processes can occur only on the surface, not within the mineralized mass. Direct addition of new bone to the surface of existing bone can and does occur through the activity of cells in the periosteum—the soft tissue membrane that covers the bone. Formation of new cells occurs in the periosteum, and extracellular material secreted there is mineralized and becomes a new bone. This process is called the *direct* or *surface apposition* of bone. Interstitial growth is a prominent aspect of overall skeletal growth because a major portion of the skeletal system is originally modeled in cartilage. This includes the basal part of the skull, as well as the trunk and limbs.

Fig. 2.19 shows the cartilaginous and neural development of the face and cranium at 8 and 12 weeks of intrauterine development. Cartilaginous skeletal development occurs most rapidly during the third month of intrauterine life. A continuous plate of cartilage extends from the nasal capsule posteriorly all the way to the foramen magnum at the base of the skull. It must be kept in mind that cartilage is a nearly avascular tissue whose internal cells are supplied by diffusion through the outer layers. This means that the cartilage must be thin. At early stages in the development of the fetus (the fetal stage begins at the start of the third month), its extremely small size makes a chondroskeleton feasible, but with further growth such an arrangement is no longer possible without an internal blood supply.

During the fourth month in utero, there is an ingrowth of blood vascular elements into various points of the chondrocranium (and the other parts of the early cartilaginous skeleton). These areas become centers of ossification, at which cartilage is transformed



• **Fig. 2.19** Development and maturation of the chondrocranium (cartilage, *light blue; bone, dark blue*). (A) Diagrammatic representation at about 8 weeks. Note that an essentially solid bar of cartilage extends from the nasal capsule anteriorly to the occipital area posteriorly. (B) Skeletal development at 12 weeks. Ossification centers have appeared in the midline cartilage structures, and in addition, intramembranous bone formation of the jaws and braincase has begun. From this point on, bone replaces the cartilage of the original chondrocranium rapidly so that only the small cartilaginous synchondroses connecting the bones of the cranial base remain.

into bone in the process called *endochondral ossification*, and islands of bone appear in the sea of surrounding cartilage (see Fig. 2.19B). The cartilage continues to grow rapidly but is replaced by bone with equal rapidity. The result is that the amount of bone increases rapidly and the relative (but not the absolute) amount of cartilage decreases. Eventually, the old chondrocranium is represented only by small areas of cartilage interposed between large sections of bone, which assume the characteristic form of the ethmoid, sphenoid, and basioccipital bones. Growth at these cartilaginous connections between the skeletal bones is like growth in the limbs.

In the long bones of the extremities, areas of ossification appear in the center of the bones and at the ends, ultimately producing a central shaft called the *diaphysis* and a bony cap on each end called the *epiphysis*. Between the epiphysis and diaphysis is a remaining area of uncalcified cartilage called the *epiphyseal plate* (Fig. 2.20A). The epiphyseal plate cartilage of the long bones is a major center for their growth, and in fact, this cartilage is responsible for almost all growth in the length of these bones. The periosteum on the surfaces of the bones also plays an important role in adding to thickness and reshaping the external contours.

Near the outer end of each epiphyseal plate is a zone of actively dividing cartilage cells. Some of these, pushed toward the diaphysis by proliferative activity beneath, undergo hypertrophy, secrete an extracellular matrix, and eventually degenerate as the matrix begins to mineralize and then is rapidly replaced by bone. If the rate at which cartilage cells proliferate is equal to or greater than the rate at which they mature, growth will continue. Eventually, however, toward the end of the normal growth period, the rate of maturation exceeds the rate of proliferation, the last of the cartilage is replaced by bone, and the epiphyseal plate disappears. At that point, the growth of the bone is complete, except for surface changes in thickness, which can be produced by the periosteum.

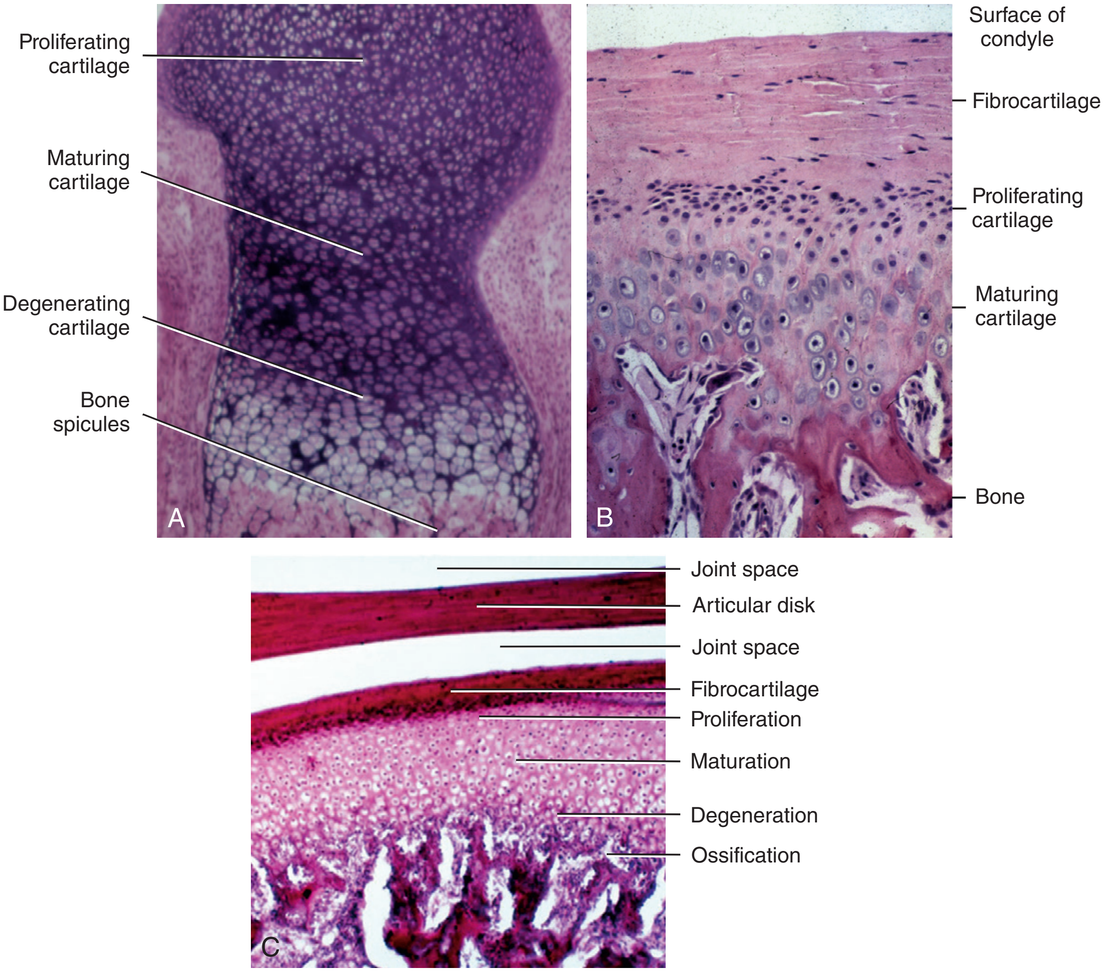
Endochondral ossification also occurs at the mandibular condyle, which superficially looks like half an epiphyseal plate (Fig. 2.20B and C). As we will see, however, the cartilage of the condyle does not behave like an epiphyseal plate—and the difference is important in understanding mandibular growth.

Not all bones of the adult skeleton were represented in the embryonic cartilaginous model, and it is possible for bone to form by secretion of bone matrix directly within connective tissues without any intermediate formation of cartilage. Bone formation of this type is called *intramembranous ossification*. This type of bone formation occurs in the cranial vault and both jaws (Fig. 2.21).

Early in embryonic life (which is discussed in some detail later at the beginning of Chapter 5), the mandible of higher animals develops in the same area as the cartilage of the first pharyngeal arch—Meckel cartilage. Unlike other bones of the cranium, the development of the mandible begins as a condensation of mesenchyme just lateral to Meckel cartilage and proceeds entirely by intramembranous bone formation (Fig. 2.22). Meckel cartilage disintegrates and largely disappears as the bony mandible develops. Remnants of this cartilage are transformed into a portion of two of the small bones that form the conductive ossicles of the middle ear but not into a significant part of the mandible. Its perichondrium persists as the sphenomandibular ligament. The condylar cartilage develops initially as an independent secondary cartilage, which is separated by a considerable gap from the body of the mandible (Fig. 2.23). Early in fetal life, it fuses with the developing mandibular ramus.

The maxilla forms initially from a center of mesenchymal condensation in the maxillary process. This area is located on the lateral surface of the nasal capsule, the most anterior part of the chondrocranium, but endochondral ossification does not contribute directly to the formation of the maxillary bone. An accessory cartilage, the zygomatic or malar cartilage, which forms in the developing malar process, disappears and is totally replaced by bone well before birth, unlike the mandibular condylar cartilage, which persists.

Whatever the location for intramembranous bone formation, interstitial growth within the mineralized mass is impossible, and



• **Fig. 2.20** (A) Endochondral ossification at an epiphyseal plate. Growth occurs by proliferation of cartilage, occurring here at the top. Maturing cartilage cells are displaced away from the area of proliferation, undergo hypertrophy, degenerate, and are replaced by spicules of bone, as seen in the bottom. (B and C) Endochondral ossification in the head of the condyle. A layer of fibrocartilage lies on the surface, with proliferating cells just beneath. Maturing and degenerating cartilage cells can be seen toward the area of ossification.

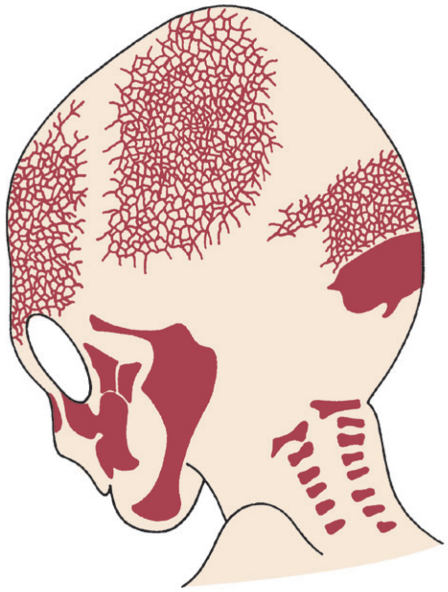
the bone must be formed entirely by apposition of new bone to free surfaces. Its shape can be changed through the removal (resorption) of bone in one area and the addition (apposition) of bone in another (see Fig. 2.15). This balance of apposition and resorption, with new bone being formed in some areas while old bone is removed in others, is an essential component of the growth process. It has two components: *modeling* and *remodeling*.

Bone modeling adapts structure to function by changing bone size and shape to maintain bone strength as the loading of the bone changes. This is mediated by the independent activities of osteoblasts and osteoblasts. This process also includes bone drift, such as the relocation of the mandibular ramus during growth (described in detail later). Bone remodeling occurs via osteocyte apposition and osteoclast resorption in the same area without changing the shape of bone. An excellent example of remodeling is the process that takes place with tooth movement, but internal remodeling of bony structures occurs in a continuous cycle.[14] Keeping this distinction between modeling and remodeling in mind can make it easier to understand the bony changes that occur during growth that are discussed in the following sections of this chapter, where these terms are *not* used interchangeably.

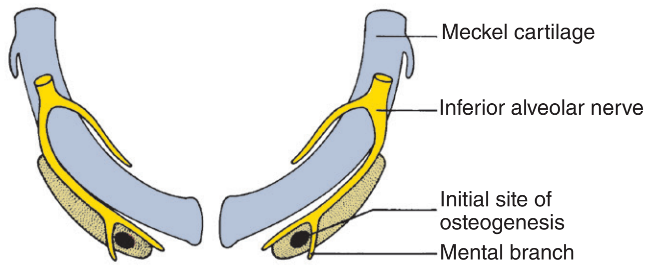
Sites and Types of Growth in the Craniofacial Complex

To understand growth in any area of the body, it is necessary to understand (1) the sites or location of growth, (2) the type of growth occurring at that location, (3) the mechanism of growth (i.e., how growth changes occur), and (4) the determinant or controlling factors in that growth.

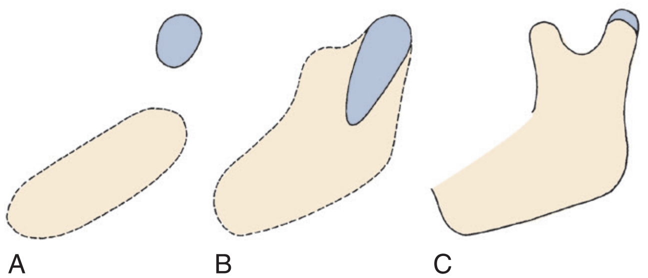
In the following discussion of sites and types of growth in the head and face, it is convenient to divide the craniofacial complex into four areas that grow rather differently: the cranial vault, the bones that cover the upper and outer surface of the brain; the cranial base, the bony floor under the brain, which also is the dividing line between the cranium and the face; the nasomaxillary complex, made up of the nose, maxilla, and associated small bones; and the mandible. The sites and types of growth are discussed in the following section of this chapter. The mechanism and determinants for growth in each area, as they are viewed from the perspective of current theories of growth control, are discussed later.



• **Fig. 2.21** The bones of the skull of a 12-week-old fetus, drawn from a cleared alizarin-stained specimen. (Redrawn from Sadler TW, Langman J. *Langman's Medical Embryology*. 9th ed. Lippincott Williams & Wilkins; 2003.)



• **Fig. 2.22** Diagrammatic representation of the relation of initial bone formation in the mandible to Meckel cartilage and the inferior alveolar nerve. Bone formation begins just lateral to Meckel cartilage and spreads posteriorly along it without any direct replacement of the cartilage by the newly forming bone of the mandible. (Redrawn from Ten Cate AR. *Oral Histology: Development, Structure, and Function*. 5th ed. Mosby; 1998.)



• **Fig. 2.23** The condylar cartilage *(blue)* develops initially as a separate area of condensation from that of the body of the mandible and is only later incorporated within. (A) Separate areas of mesenchymal condensation at 8 weeks. (B) Fusion of the cartilage with the mandibular body at 4 months. (C) Situation at birth (reduced to scale).

Cranial Vault

The cranial vault is made up of a number of flat bones that are formed directly by intramembranous bone formation without cartilaginous precursors. From the time that ossification begins at several locations that foreshadow the eventual anatomic bony units, the growth process is entirely the result of periosteal activity at the surfaces of the bones. Modeling (addition of new bone) and growth occur primarily at the periosteum-lined contact areas between adjacent skull bones, the *cranial sutures*, but periosteal activity also produces modeling changes on the inner and outer surfaces of these platelike bones.

At birth, the flat bones of the skull are rather widely separated by loose connective tissues (Fig. 2.24). These open spaces, the fontanelles, allow a considerable amount of deformation of the skull at birth. This is important in allowing the relatively large head to pass through the birth canal (see Chapter 3 for more detail). After birth, the apposition of bone along the edges of the fontanelles eliminates these open spaces quickly, but the bones remain separated by a thin, periosteum-lined suture for many years, eventually fusing in adult life.

Despite their small size, the apposition of new bone at these sutures is the major mechanism for growth of the cranial vault. Although the majority of the growth in the cranial vault occurs at the sutures, there is a tendency for the bone to be removed from the inner surface of the cranial vault, while at the same time new bone is added on the exterior surface. This modeling of the inner and outer surfaces allows for changes in contour during growth.

Cranial Base

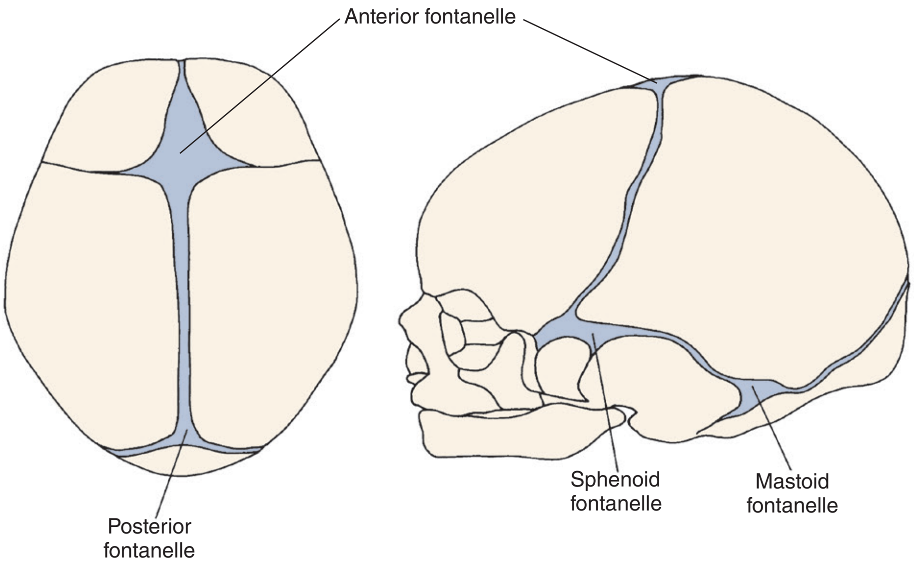
In contrast to the cranial vault, the bones of the base of the skull (the cranial base) are formed initially in cartilage, and these cartilage models are later transformed into bone by endochondral ossification. The situation is more complicated, however, than in a long bone with its epiphyseal plates. The cartilage modeling is particularly true of the midline structures. As one moves laterally, growth at sutures and surface remodeling becomes more important.

As indicated previously, ossifications appear early in embryonic life in the chondrocranium, indicating the eventual location of the basioccipital, sphenoid, and ethmoid bones that form the cranial base. As ossification proceeds, bands of cartilage called *synchondroses* remain between the centers of ossification (Fig. 2.25). These important growth sites (and centers) are the synchondrosis between the sphenoid and occipital bones, or *spheno-occipital synchondrosis;* the *intersphenoid synchondrosis* between two parts of the sphenoid bone; and the *spheno-ethmoidal synchondrosis* between the sphenoid and ethmoid bones. Histologically, a synchondrosis looks like a two-sided epiphyseal plate (Fig. 2.26). Synchondrosis has an area of cellular hyperplasia in the center with bands of maturing cartilage cells extending in both directions, which will eventually be replaced by bone.

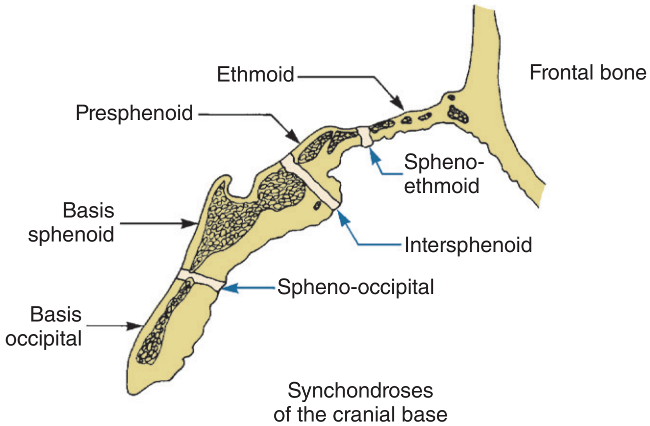
A significant difference from the bones of the extremities is that immovable joints develop between the bones of the cranial base, in considerable contrast to the highly movable joints of the extremities. The cranial base is thus rather like a single long bone, except that there are multiple epiphyseal plate–like synchondroses. Immovable joints also occur between most of the other cranial and facial bones, the mandible being the only exception. The periosteum-lined sutures of the cranium and face, containing no cartilage, are quite different from the cartilaginous synchondroses of the cranial base.

Maxilla (Nasomaxillary Complex)

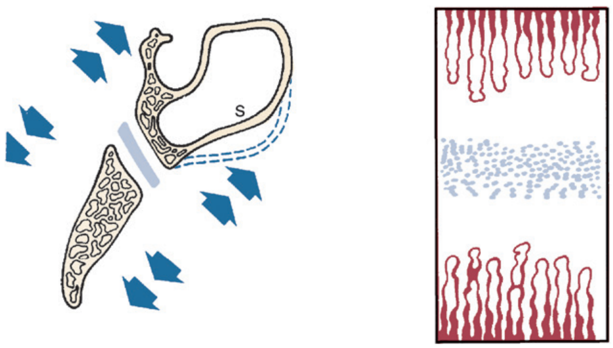
The maxilla develops postnatally entirely by intramembranous ossification. Because there is no cartilage replacement, growth



• **Fig. 2.24** The fontanelles of the newborn skull *(blue)*.



• **Fig. 2.25** Diagrammatic representation of the synchondroses of the cranial base, showing the locations of these important growth sites.



• **Fig. 2.26** Diagrammatic representation of growth at the spheno-occipital synchondrosis. A band of immature proliferating cartilage cells is located at the center of the synchondrosis, a band of maturing cartilage cells extends in both directions away from the center, and endochondral ossification occurs at both margins. Growth at the synchondrosis lengthens this area of the cranial base. Even within the cranial base, bone remodeling on surfaces is also important—it is also the mechanism by which the sphenoid sinus(es) enlarges, for instance.

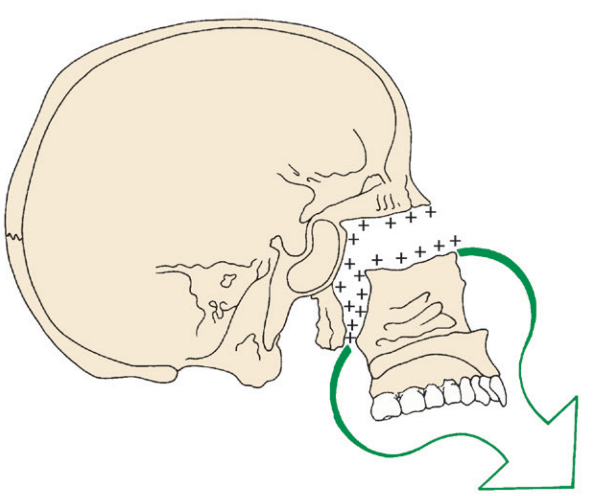
occurs in two ways: (1) by apposition of bone at the sutures that connect the maxilla to the cranium and cranial base and (2) by surface modeling. In contrast to the cranial vault, however, surface changes in the maxilla are quite dramatic and as important as changes in the sutures. In addition, the maxilla is moved forward by the growth of the cranial base behind it.

The growth pattern of the face requires that it grow "out from under the cranium," which means that as it grows, the maxilla must move a considerable distance downward and forward relative to the cranium and cranial base. This is accomplished in two ways: (1) by a push from behind created by cranial base growth and (2) by growth at the sutures. Because the maxilla is attached to the anterior end of the cranial base, lengthening of the cranial base pushes it forward. Up until about age 6, displacement from cranial base growth is an important part of the maxilla's forward growth. Failure of the cranial base to lengthen normally, as in achondroplasia (see Fig. 5.27) and several congenital syndromes, creates a characteristic midface deficiency. At about age 7, cranial base growth stops, and then sutural growth is the only mechanism for bringing the maxilla forward.

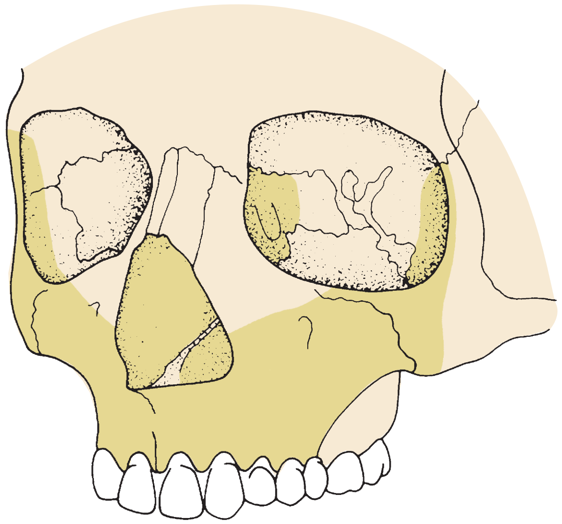
As Fig. 2.27 illustrates, the sutures attaching the maxilla posteriorly and superiorly are ideally situated to allow its downward and forward repositioning. As the downward and forward movement occurs, the space that would otherwise open at the sutures is filled in by the proliferation of bone at these locations. The sutures remain the same width, and the various processes of the maxilla become longer. Bone apposition occurs on both sides of a suture, so the bones to which the maxilla is attached also become larger. Part of the posterior border of the maxilla is a free surface in the tuberosity region. Bone is added at this surface, creating additional space into which the primary and then the permanent molar teeth successively erupt.

It is interesting to note that as the maxilla grows downward and forward, its front surfaces are modeled, and bone is removed from most of the anterior surface. Note in Fig. 2.28 that almost the entire anterior surface of the maxilla is an area of resorption, not apposition.

To understand this seeming paradox, it is necessary to comprehend that two quite different processes are going on simultaneously.



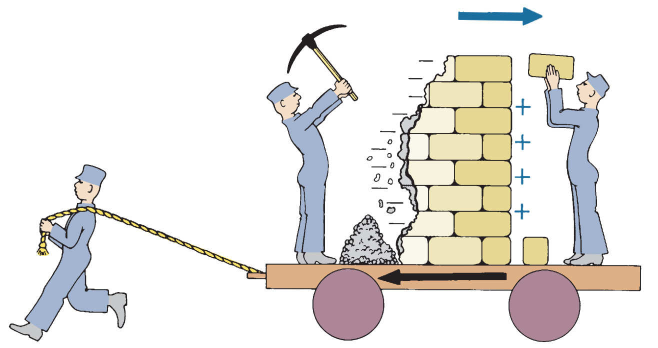
• **Fig. 2.27** As the growth of surrounding soft tissues translates the maxilla downward and forward, opening up space at its superior and posterior sutural attachments, new bone is added on both sides of the sutures. (Redrawn from Enlow DH, Hans MG. *Essentials of Facial Growth*. WB Saunders; 1996.)



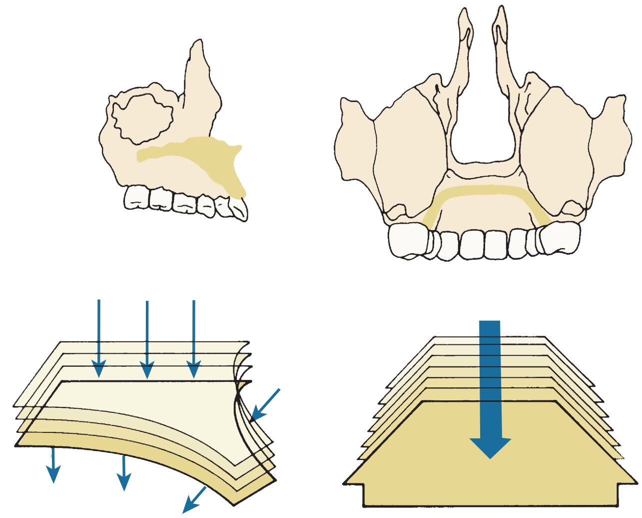
• **Fig. 2.28** As the maxilla is carried downward and forward, its anterior surface tends to resorb. Resorption surfaces are shown here in dark yellow. Only a small area around the anterior nasal spine is an exception. (Redrawn from Enlow DH, Hans MG. *Essentials of Facial Growth*. WB Saunders; 1996.)

The overall growth changes are the result of both a downward and forward translation of the maxilla and simultaneous surface modeling. The whole bony nasomaxillary complex is moving downward and forward relative to the cranium, being translated in space. Enlow, whose careful anatomic studies of the facial skeleton underlie much of our present understanding, illustrated this in cartoon form (Fig. 2.29). The maxilla is like the platform on wheels, being rolled forward, while at the same time, its surface, represented by the wall in the cartoon, is being reduced on its anterior side and built up posteriorly, moving in space opposite to the direction of overall growth.

It is not necessarily true that modeling changes oppose the direction of translation. Depending on the specific location, translation and modeling may either oppose each other or produce an additive effect. The effect is additive, for instance, on the roof of



• **Fig. 2.29** Surface modeling of a bone in the opposite direction to that in which it is being translated by the growth of adjacent structures creates a situation analogous to this cartoon, in which the wall is being rebuilt to move it backward at the same time the platform on which it is mounted is being moved forward. (Redrawn from Enlow DH, Hans MG. *Essentials of Facial Growth*. WB Saunders; 1996.)

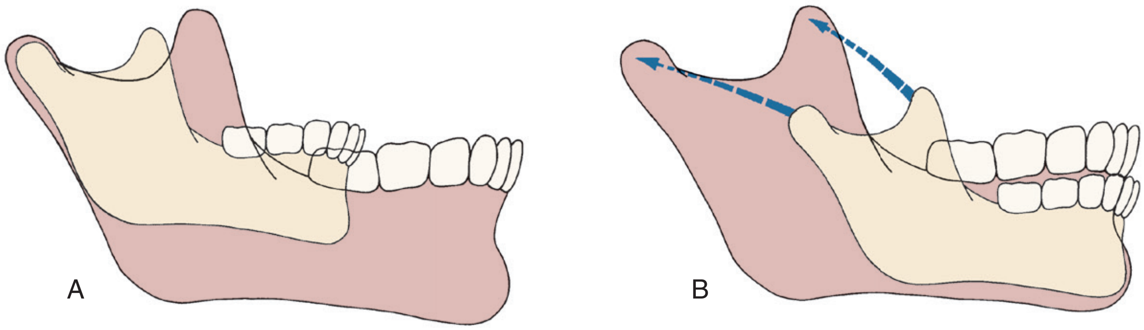


• **Fig. 2.30** Modeling of the palatal vault (which is also the floor of the nose) moves it in the same direction as it is being translated; bone is removed from the floor of the nose and added to the roof of the mouth. On the anterior surface, however, bone is removed, partially canceling the forward translation. As the vault moves downward, the same process of bone remodeling also widens it. (Redrawn from Enlow DH, Hans MB. *Essentials of Facial Growth*. WB Saunders; 1996.)

the mouth. This area is carried downward and forward along with the rest of the maxilla, but at the same time, bone is removed on the nasal side and added on the oral side, thus creating an additional downward and forward movement of the palate (Fig. 2.30). Immediately adjacently, however, the anterior part of the alveolar process is a resorptive area, so removal of bone from the surface here tends to cancel some of the forward growth that otherwise would occur because of translation of the entire maxilla.

Mandible

In contrast to the maxilla, both endochondral and periosteal activity are important in the growth of the mandible, and displacement created by cranial base growth that moves the temporomandibular joint plays a negligible role (with rare exceptions; see Fig. 4.10). Cartilage covers the surface of the mandibular condyle at the



• **Fig. 2.31** (A) Growth of the mandible, as viewed from the perspective of a stable cranial base. The chin moves downward and forward. (B) Mandibular growth, as viewed from the perspective of vital staining studies, which reveal minimal changes in the body and chin area, while there is exceptional growth and modeling of the ramus, moving it posteriorly. The correct concept is that the mandible is translated downward and forward and grows upward and backward in response to this translation, maintaining its contact with the skull.

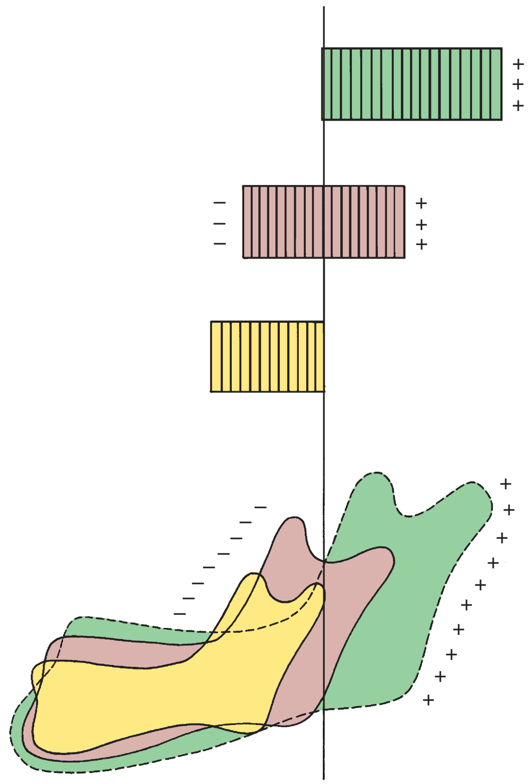
temporomandibular joint. Although this cartilage is not like the cartilage at an epiphyseal plate or a synchondrosis, hyperplasia, hypertrophy, and endochondral replacement do occur there. All other areas of the mandible are formed and grow by direct surface apposition (modeling).

The overall pattern of growth of the mandible can be represented in two ways, as shown in Fig. 2.31. Depending on the frame of reference, both are correct. If the cranium is the reference area, the chin moves downward and forward. On the other hand, if data from vital staining experiments are examined, it becomes apparent that the principal sites of growth of the mandible are the posterior surface of the ramus and the condylar and coronoid processes. There is little change along the anterior part of the mandible. From this frame of reference, Fig. 2.31B is the correct representation.

As a growth site, the chin is almost inactive. It is translated downward and forward, as the actual growth occurs at the mandibular condyle and along the posterior surface of the ramus. The body of the mandible grows longer by periosteal apposition of bone only on its posterior surface, while the ramus grows higher by endochondral replacement at the condyle accompanied by surface modeling. Conceptually, it is correct to view the mandible as being translated downward and forward while increasing in size by growing upward and backward. The translation occurs largely as the bone moves downward and forward along with the soft tissues in which it is embedded.

Nowhere is there a better example of modeling resorption than the backward movement of the ramus of the mandible. The mandible grows longer by apposition of new bone on the posterior surface of the ramus. At the same time, large quantities of bone are removed from the anterior surface of the ramus (Fig. 2.32). In essence, the body of the mandible grows longer as the ramus moves away from the chin, and this occurs by the removal of bone from the anterior surface of the ramus and the deposition of bone on the posterior surface. Based on this, what was the posterior surface at one time becomes the center at a later date and eventually may become the anterior surface as modeling proceeds.

In infancy the ramus is located at about the spot where the primary first molar will erupt. Progressive posterior modeling creates space for the second primary molar and then for the sequential eruption of the permanent molar teeth. More often than not, however, this growth ceases before enough space has been created for the eruption of the third permanent molar, which becomes impacted in the ramus.



• **Fig. 2.32** As the mandible grows in length, the ramus is extensively modeled, so much so that bone at the tip of the condylar process at an early age can be found at the anterior surface of the ramus some years later. Given the extent of surface remodeling changes, it is an obvious error to emphasize endochondral bone formation at the condyle as the major mechanism for growth of the mandible. (Redrawn from Enlow DH, Hans MB. *Essentials of Facial Growth*. WB Saunders; 1996.)

Further aspects of the growth of the jaws, especially in relation to the timing of orthodontic treatment, are discussed in Chapter 4.

Facial Soft Tissues

An important concept is that the growth of the facial soft tissues does not perfectly parallel the growth of the underlying hard tissues. Let us consider the growth of the lips and nose in more detail.

Growth of the Lips

The lips trail behind the growth of the jaws before adolescence, then undergo a growth spurt to catch up. Because lip height is relatively short during the mixed dentition years, lip separation at rest (often termed *lip incompetence*) is maximal during childhood and decreases during adolescence (Fig. 2.33). Lip thickness reaches its maximum during adolescence, then decreases (Fig. 2.34)—to the point that in their 20s and 30s, some people may consider the loss of lip thickness a problem and seek treatment to increase it. This is discussed further in Chapter 7.

Growth of the Nose

Growth of the nasal bone is complete at about age 10. Growth thereafter is only of the nasal cartilage and soft tissues, both of which undergo a considerable adolescent spurt. The result is that the nose becomes much more prominent in adolescence, especially in boys (Fig. 2.35). The lips are framed by the nose above and chin below, both of which become more prominent with adolescent and postadolescent growth, while the lips do not, so the relative prominence of the lips decreases. This can become an

• **Fig. 2.33** Growth of the lips trails behind growth of the facial skeleton until puberty, then catches up and tends to exceed skeletal growth thereafter. As a result, lip separation and exposure of the maxillary incisors is maximal before adolescence and decreases during adolescence and early adult life. (A) Age 11 years, 9 months, prior to puberty. (B) Age 14 years, 8 months, after the adolescent growth spurt. (C) Age 16 years, 11 months. (D) Age 18 years, 6 months.

important point in determining how much lip support should be provided by the teeth at the time orthodontic treatment typically ends in late adolescence.

Changes in the facial soft tissues with aging, which also must be taken into consideration in planning orthodontic treatment, are covered in Chapter 4.

Theories of Growth Control

It is true that growth is strongly influenced by genetic factors, but it also can be significantly affected by the environment in the form of nutritional status, degree of physical activity, health or illness, and a number of similar factors. Because a major part of the need for orthodontic treatment is created by disproportionate growth of the jaws, to understand the etiologic processes of malocclusion and dentofacial deformity, it is necessary to learn how facial growth is influenced and controlled. Great strides have been made in recent years in improving the understanding of growth control. Exactly what determines the growth of the jaws, however, remains unclear and continues to be the subject of intensive research.

Three major theories in recent years have attempted to explain the determinants of craniofacial growth: (1) bone, like other

• **Fig. 2.34** Lip thickness increases during the adolescent growth spurt and then decreases (and therefore is maximal at surprisingly early ages). For some girls, loss of lip thickness is perceived as a problem in their early 20s. (A) Age 11 years, 9 months, prior to puberty. (B) Age 14 years, 8 months, after the adolescent growth spurt. (C) Age 16 years, 11 months. (D) Age 18 years, 6 months.

tissues, is the primary determinant of its own growth; (2) cartilage is the primary determinant of skeletal growth, while bone responds secondarily and passively; and (3) the soft tissue matrix in which the skeletal elements are embedded is the primary determinant of growth, and both bone and cartilage are secondary followers.

The major difference in the theories is the location at which genetic control is expressed. The first theory implies that genetic control is expressed directly at the level of the bone; therefore its locus should be the periosteum. The second, or cartilage, theory suggests that genetic control is expressed in the cartilage, while bone responds passively to being displaced as cartilage grows. Indirect genetic control, whatever its source, is called *epigenetics*. The third theory assumes that genetic control is mediated to a large extent outside the skeletal system and that the growth of both bone and cartilage is controlled epigenetically, occurring only in response to a signal from other tissues. In contemporary thought, the truth is to be found in some synthesis of the second and third theories; the first, although it was the dominant view until the 1960s, has largely been discarded.

Level of Growth Control: Sites Versus Centers of Growth

Distinguishing between a *site* of growth and a *center* of growth clarifies the differences between the theories of growth control.

• **Fig. 2.35** The nasal bone grows up until about age 10, but after age 10, growth of the nose is largely in the cartilaginous and soft tissue portions. Especially in boys, the nose becomes much more prominent as growth continues after the adolescent growth spurt (and this process continues into the adult years). (A) Age 4 years, 9 months. (B) Age 12 years, 4 months. (C) Age 14 years, 8 months. (D) Age 17 years, 8 months.